Medical Lib.

NOV 15 1928

VOLUME 36 NUMBER 5

AMERICAN JOURNAL

OF

DISEASES OF CHILDREN

EDITORIAL BOARD

CLIFFORD G. GRULEE, Chicago

OSCAR M. SCHLOSS, New York
FRITZ B. TALBOT, Boston
H. F. HELMHOLZ, Rochester, Minn.
W. McKIM MARRIOTT, St. Louis
L. R. DeBUYS, New Orleans
JOHN C. GITTINGS, Philadelphia

NOVEMBER, 1928

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $5.00

Entered as Second-Class Matter, January 4, 1911, at the Postoffice at Chicago, Illinois, Under Act of Congress of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on June 14, 1918.

CONTENTS OF PREVIOUS NUMBER

OCTOBER 1928. NUMBER 4

Self Selection of Diet by Newly Weaned Infants: An Experimental Study. Clara M. Davis, M.D., Chicago.

Communicating Hydrocephalus: So-Called Idiopathic Hydrocephalus. Joseph H. Globus, M.D., New York.

Initial Fever in Tuberculosis. Arvid Wallgren, M.D., Göteborg, Sweden.

Calcium and Phosphorus Metabolism of Epileptic Children Receiving a Ketogenic Diet. Martha Van Kirk Nelson, M.S., Iowa City.

Studies in Immunity: I. Nonspecific Factors Influencing the Reaction of the Skin to Tuberculin. A. Graeme Mitchell, M.D.; William B. Wherry, M.D.; Bernice Eddy, Ph.D., and Frank E. Stevenson, M.D., Cincinnati.

Blood in the Stools of the New-Born. Barnet E. Bonar, M.D., Salt Lake City.

Seasonal Variation of the Antirachitic Effect of Sunshine: II. Frederick F. Tisdall, M.D. (Tor.), and Alan Brown, M.B. (Tor.), Toronto.

The Respiratory Metabolism in Infancy and in Childhood: X. The Respiratory Exchange in Marasmus; Specific Dynamic Action of Food in Normal and in Marasmic Infants. Samuel Z. Levine, M.D.; James R. Wilson, M.D., and Gertrude Gottschall, B.S., New York.

The Distribution of the Globulin and Albumin Fractions in the Blood and in the Urine in Nephrosis: A Preliminary Report. F. W. Schlutz, M.D.; W. W. Swanson, M.D., and M.R. Ziegler, Ph.D., Minneapolis.

Case Reports:
- Intra-Uterine Lobar Pneumonia and Pneumococcemia. Abraham S. Gordon, M.D., and Max Lederer, M.D., Brooklyn.
- Fetal Peritonitis: Report of a Case Following Spontaneous Rupture of the Large Intestine. Alfred E. Fischer, M.D., New York.
- Pilonidal Sinus as a Route of Infection in a Case of Staphylococcus Meningitis. Warren Ripley, M.D., and D. Clark Thompson, M.D., Montclair, N. J.
- Temporary Amaurosis Complicating Pertussis. Abraham Litvak, M.D., Brooklyn.
- Premature Infant, Birth Weight 680 Grams, with Survival. D. S. Pulford, M.D., and W. J. Blevins, M.D., Woodland, Calif.

Progress in Pediatrics:
- Cerebrospinal Fluid in Infants and in Children. A. Levinson, M.D., Chicago.

Pediatric Biographies:
- Hieronymous Mercurialis. John Ruhräh, Baltimore.

News and Comment.

Abstracts from Current Literature.

Society Transactions:
- Chicago Pediatric Society.
- North Pacific Pediatric Society.

Book Reviews.

Directory of Pediatric Societies.

COPYRIGHT, 1928, BY THE AMERICAN MEDICAL ASSOCIATION

American Journal of Diseases of Children

VOLUME 36 NOVEMBER, 1928 NUMBER 5

HEALING IN INFANTILE SCURVY AS SHOWN BY X-RAY *

STAFFORD McLEAN, M.D.
NEW YORK
AND
RUSTIN McINTOSH, M.D.
BALTIMORE

Any advance in the knowledge of a disease as widely known as scurvy can be achieved only by first studying the results of the painstaking accumulation of facts made by a host of observers. By pyramiding the roentgenologic investigations of others and using this as a base line for further study, the task of amplifying the information concerning the osseous lesions in this disease has been made easier for us.

Although numerous contributions to the literature of infantile scurvy have appeared in recent years, and among them several which deal mainly with its roentgenologic manifestations, they have for the most part been concerned largely with some particular phase of the disease. Thus, Pelkan,[1] Wimberger,[2] Néchitch,[3] and Baetjer [4] have considered the problem of differential diagnosis in acute scurvy. Wimberger,[5] Frank,[6] and, more recently, Schwartz,[7] have stressed the late manifestations and retrospective diagnosis. In his monograph in 1908, Fraenkel [8] laid relatively little emphasis on the time element in the changing picture of the healing process, and Bromer's [9] recent paper, published this year, is devoted mainly to differential diagnosis.

* Received for publication, June 20, 1928.

* From the Babies' Hospital and Department of Diseases of Children, College of Physicians and Surgeons, Columbia University, New York.

1. Pelkan, K. F.: The Roentgenogram in Early Scurvy, Am. J. Dis. Child. **30**:174 (Aug.) 1925.

2. Wimberger, H.: Ztschr. f. Kinderh. **36**:279, 1923; Fortschr. a. d. Geb. d. Roentgenstrahlen **32**:17, 1924; Ergebn. d. inn. med. u. Kinderh. **28**:288, 1925.

3. Néchitch, A. R.: Scorbut infantile, Thesis, Univ. Paris, 1924.

4. Baetjer, F. H.: Differential Diagnosis by X-Ray between Rickets, Syphilis, Scurvy, and Condition known as Perthé's Disease, M. Clin. N. Amer. **2**:1703 (May) 1919.

5. Wimberger (footnote 2, third reference).

6. Frank, H. M.: Ztschr. f. Kinderh. **27**:127, 1920.

7. Schwartz, A. B.: The Epiphyseal Changes in the Diagnosis of Scurvy, Am. J. Dis. Child. **34**:765 (Nov.) 1927.

8. Fraenkel, E.: Fortschr. a. d. Geb. d. Roentgenstrahlen, 1908, vol. 18.

9. Bromer, R. S.: The Roentgen-Ray Diagnosis of Infantile Scurvy, Am. J. Roentgenol. **19**:112 (Feb.) 1928.

Our study of healing in infantile scurvy is based on 115 roentgenographic examinations made in 52 cases at various stages of the disease, in some instances ranging over a period of more than eleven years after the active phase of the disease. It has been our aim to observe the order and rate of change of the various manifestations of the healing process as they appeared in the x-ray and to determine some of the factors, aside from the question of dietary adequacy, which influence this rate. In all cases, both anteroposterior and lateral views of the lower extremities were used, and when positive appearances were noted in one view and not in another, they were recorded as positive for that examination. The upper extremities were studied only when symptoms indicated the presence of lesions there, and these results have been included in the figures reported; but in two cases in which there were manifest subperiosteal hemorrhages in the skull, and in one involving the scapulae, these films have not been included. Owing to the difficulty in holding infants still, double intensifying screens were used and the length of exposure kept down to a minimum, ordinarily about one tenth of a second. While screening necessarily sacrifices detail to a certain degree, particularly in the trabecular structure, the advantage of increased speed is paramount in working with infants and children.

When we undertook to study the x-ray evidence of healing, we were at once confronted with difficulty in answering the fundamental questions. When does healing commence, and what are the criteria of activity? The great variability in x-ray manifestations in individual cases at the time when the diagnosis of infantile scurvy is first made soon forced us to the conclusion that certain infants may show evidence of healing in the x-ray even though the disease is clinically active on admission to the hospital. It is readily understandable that healing, occurring in short spurts and at times accompanied by improvement in clinical symptoms, may result from a fortuitous "error" in a scorbutogenic diet by which the infant obtains an appreciable amount of the required vitamin. This is particularly apt to occur in cases in which the symptoms are mild, being limited perhaps to a degree of tenderness which can plausibly be ascribed to some other cause—a sufficiently common experience; so that by no means all of the patients reach the hospital with an uninterrupted progression of symptoms. Since this question of activity is so important for our purposes, it might have been hoped that the history in each case could serve to separate the unquestionably active cases from those in which some healing might have occurred before the patient came to our hands. However, histories of patients with scurvy are often unreliable, for the reason that in this era, and in New York, at least, it is a disease not of poverty but

of ignorance; and we were not inclined to rely on them for a distinction of this nicety. Neither was it possible to determine the inception of healing in all cases by noting the time of appearance in the x-rays of some unequivocal sign or evidence of a return to normal metabolism of bone. The most concise and definite roentgenographic sign of healing is probably the first appearance of lime salts in the elevated periosteum surrounding a subperiosteal hemorrhage. But since not

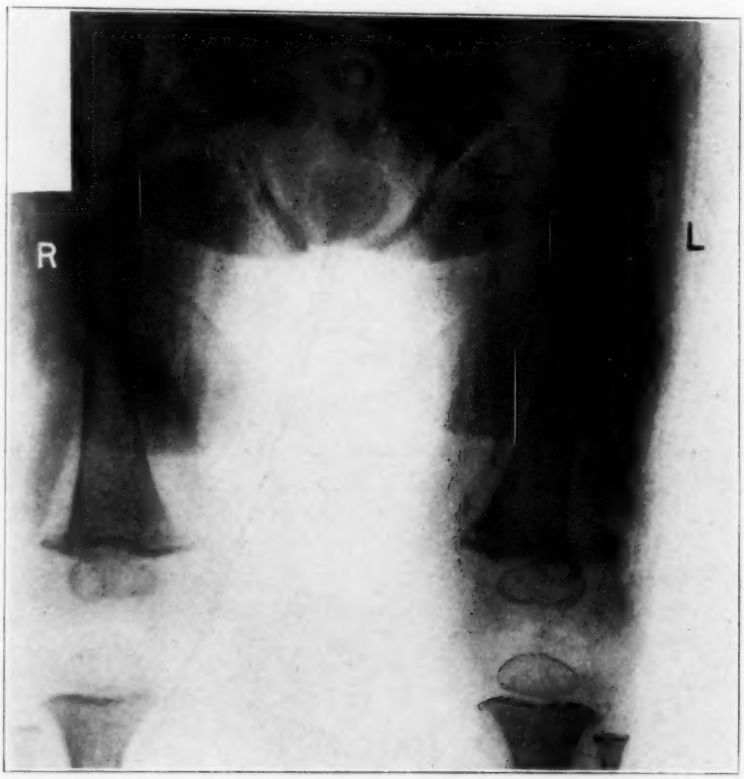

Fig. 1.—J. M., age on admission, 1 year. Pain in legs, two weeks; sore gums, ten days. Had had orange juice "a few times." Typical scorbutic attitude with tenderness of extremities; swelling of left thigh; miliaria; hemorrhages in gums; temperature, 103 F. The roentgenogram, taken ten days after admission, shows the sharp outline of the periphery of the centers of ossification, with central rarefaction; it also shows the intensity of the zone of preparatory calcification in the tibia and fibula; the faint zone of rarefaction in the tibia, extending only part way across the metaphysis; the spurs on the tibia and fibula; lateral separation of the femoral epiphysis; early calcification of a large subperiosteal hemorrhage in the femur and to a lesser extent in the tibia.

all of our cases showed such hemorrhages, since clinical improvement antedates the occurrence of this sign by several days, and since, moreover, several patients when first seen showed well advanced calcification of elevated periosteum as well as other signs of healing, we were obliged to resort to some other standard of grouping for analysis and have tabulated our cases according to their condition when the clinical diagnosis of scurvy was first made. For the purpose of our study, therefore, we

Fig. 2 (J. M.).—Condition seventeen days after admission, showing zone of rarefaction and shadows of elevated periosteum which are more conspicuous. The irregularities in the outline of the periosteal shadow should be noted.

have taken as the zero point the day on which supervised treatment was commenced, the routine for all ages being the administration of 3 ounces (90 cc.) of raw orange juice a day during the patient's stay in the hospital and thereafter the juice of one-half to a whole orange or more daily throughout the period of infancy. This regimen, except in cases showing epiphyseal separation, usually effected a complete relief of tenderness in less than a week and an improvement in other signs and

symptoms, depending to a large extent on the severity in each instance. The majority of our patients showed clinical evidence of rickets as well as scurvy.

A tabular survey of roentgenographic evidence of scurvy made in this way evades, then, the question of the first appearance of healing, though it may at the same time be held to deal adequately with the picture of uninterrupted progressive healing from the time when treat-

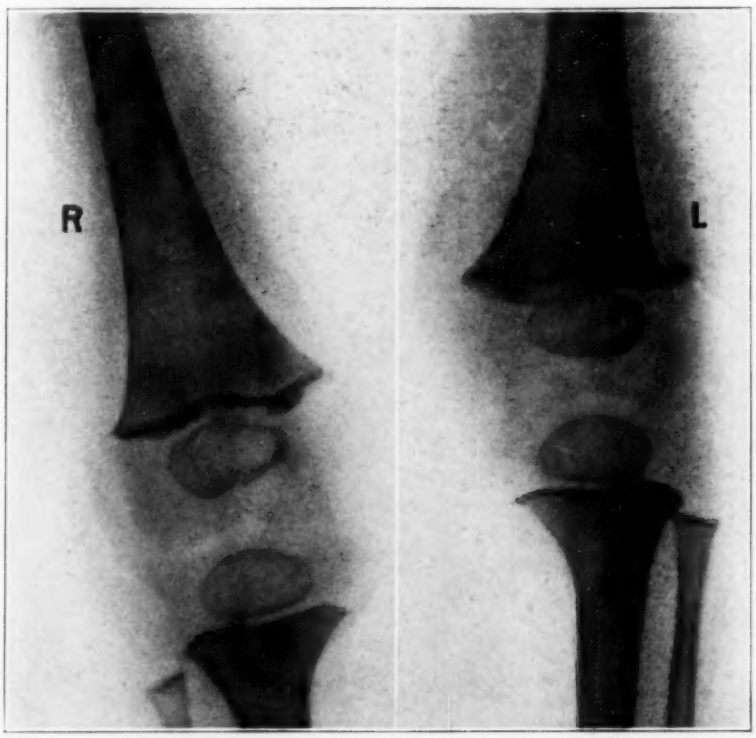

Fig. 3.—E. S., age on admission, 1 year. Tender legs, nine days; sore gums, one week. Typical scorbutic attitude with tenderness of lower extremities; swelling of left thigh; miliaria; hemorrhages around upper incisor teeth; temperature, 103.6 F. The roentgenogram, taken one day after admission, shows the sharp outline of periphery of centers of ossification, with central rarefaction; intensity of zone of preparatory calcification. The zone of rarefaction is not distinguishable. Spur formation in the femur and blurring of the trabecular markings are present.

ment was commenced. What is probably of equal importance from the clinical standpoint is the analysis which it permits of the various guises under which infantile scurvy may appear when a case first presents itself for diagnosis. We have included all grades and degrees

Figure 4

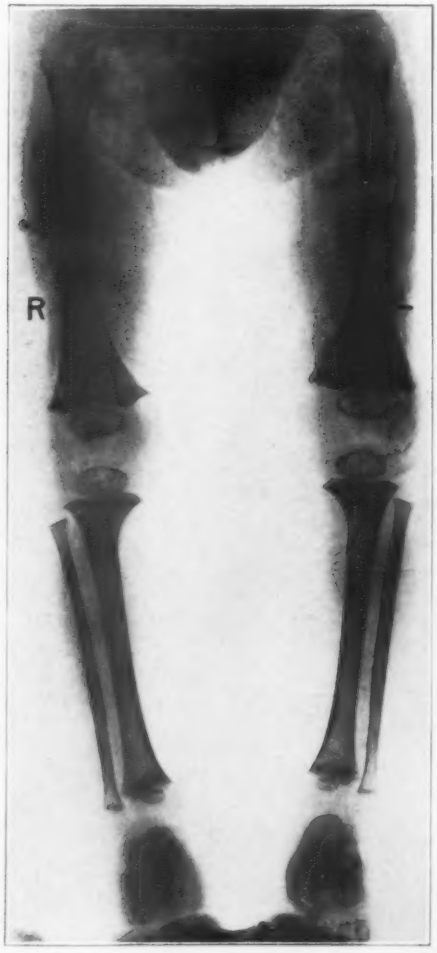

Figure 5

Fig. 4 (E. S.).—Condition eighteen days after admission, showing streaked center of ossification; zone of rarefaction conspicuous in the femur and calcification of the elevated periosteum.

Fig. 5 (E. S.).—Condition thirty-six days after admission, showing increase in the density of the entire bone shadow and double line of shadow at the end of the tibia, representing a normal zone of preparatory calcification and the old scorbutic zone which has passed into the interior of the bone. The shadow of the new bone laid down by the elevated periosteum is smaller and more dense.

Figure 6

Figure 7

Fig. 6 (E. S.).—Condition five and one half months after admission. Growth of the centers of ossification has produced a broad, dense, scalloped border with a central rarefied area traversed by coarse trabeculae. Three sets of transverse calcified lines are visible in the femur, the distal one representing the period of active scurvy. Between this and the present zone of preparatory calcification, the bone is dense with fine grained trabeculae. In the older portion of the bone, coarse trabeculae persist. The difference in rate of growth in the tibia and femur should be noted.

Fig. 7.—P. D., age on admission, 10 months. Tenderness of lower extremities, three weeks. Typical scorbutic attitude, with tenderness of both lower extremities; swelling of right knee; hemorrhage around one tooth; miliaria; temperature, 102 F. Serum calcium, 10.5 mg. in 100 cc.; phosphorus, 4 mg. in 100 cc. The roentgenogram, taken one day after admission, shows: rarefaction of all bones; sharp outline of the centers of ossification; conspicuous zone of preparatory calcification and adjacent zone of rarefaction; spur formation; comminution of the upper ends of the tibiae. The trabecular markings here are quite plain.

of the disease as they came to us, from those which according to the history, clinical picture and initial x-ray examination gave unquestionable evidence of an uninterrupted advance of the disease process to the other extreme of a patient who had been admitted to the wards of another hospital for "anterior poliomyelitis" several weeks before we saw her and while there, on hospital fare, had shown an unexpected improvement in symptoms.

Figure 8

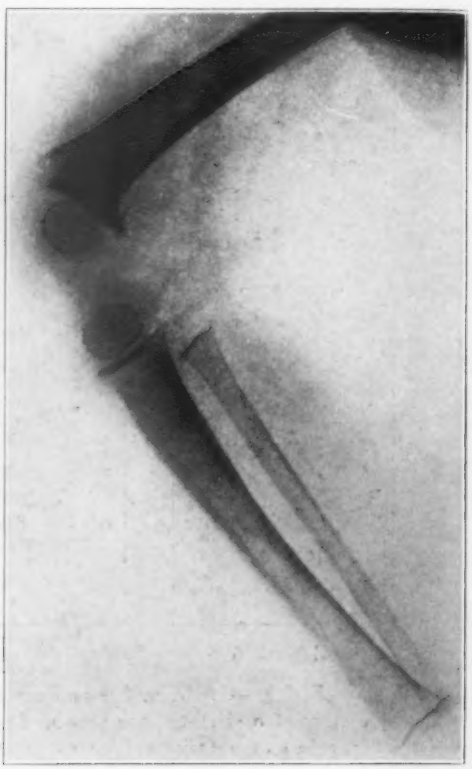

Figure 9

Fig. 8 (P. D.).—Lateral view of left leg taken on same date as figure 7. Here a posterior separation of the lower epiphysis of the femur is visible. A buckling fracture of the upper end of the tibia and spur formation at the lower ends of the tibia and fibula are present.

Fig. 9 (P. D.).—Lateral view of right leg, taken on same date as figure 7. Posterior separation of the lower epiphysis of the femur and buckling fracture of the upper end of the tibia and fibula are present.

SIGNS OF INFANTILE SCURVY SHOWN IN ROENTGENOGRAMS

The signs by which infantile scurvy may be manifest in a roentgenogram of the long bones taken early in the disease or at a period when treatment has not effected a clinical cure have been enumerated

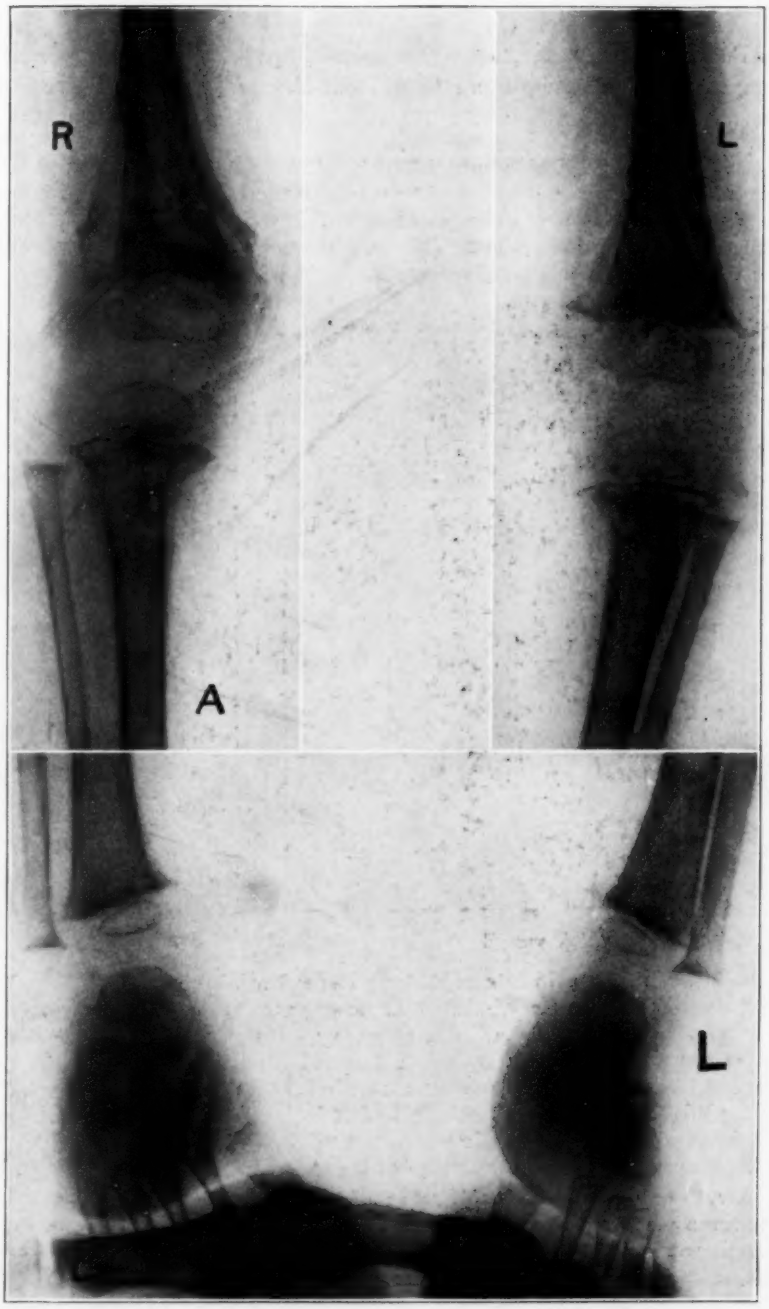

Fig. 10 (P. D.).—*A*, condition four weeks after admission, showing increased definition of trabecular markings. The zone of rarefaction is still sharply outlined in the tibiae. Shadow shows new bone laid down by the elevated periosteum. Subperiosteal fractures in the tibiae and right fibula do not cause special callus formation. *B*, detail from same film.

by Pelkan, to whose terminology we shall in part adhere in our description, in part borrowing expressions from Bromer's paper. These signs are (figs. 3, 7, 8, 9 and others):

1. A finely irregular, broadened, intensely calcified zone of preparatory calcification at the epiphyseal end of long bones, most marked where growth of bone in length is most rapid—the "white line of scurvy" in the negative, which, of course, appears black in positive prints. This constitutes that portion of the epiphyseal line which is visible in x-ray photographs.

Fig. 11 (P. D.).—Lateral view of left leg, taken on same date as figure 10.

2. A small spur at the lateral edge of the epiphysis.

3. A zone of rarefaction, referred to by Pelkan as the scurvy line and by Bromer as the framework marrow zone, immediately back of (that is, in the direction of the diaphysis) the zone of preparatory calcification, which represents lack of lime salt deposit in the newly formed abnormal spongiosa.

4. A broad, finely irregular edge of dense shadow in the epiphyseal center of ossification.

5. A rarefaction of the central portion of the center of ossification, corresponding in pathogenesis to the zone of rarefaction in the metaphysis.

6. Separation of the epiphysis.

7. A ground-glass-like transparency of the shaft, with clouding or obliteration of the trabecular structure visible in normal bone or in rickets.

8. A thinning of the cortical shadow at least to a diameter of less than one-fifth that of the shaft of the bone, sometimes to such a degree that it is represented in the negative merely by a narrow white line.

Another sign which may be sought for in the roentgenogram of a patient with acute scurvy is evidence of hemorrhage into the soft parts. This, however, involves serious difficulties of interpretation, of which

Fig. 12 (P. D.).—Lateral view of right leg, taken on same date as figure 10.

more will be mentioned subsequently. One also may occasionally find subperiosteal fractures in the ends of the diaphyses.

Pelkan stated that all of the foregoing signs will by no means be found in the roentgenogram of every patient with acute scurvy, but that in the absence of all other positive signs the presence of increased density of the zone of preparatory calcification and of the peripheral shadow of the center of ossification, together with blurring of the trabeculae, constitutes sufficient evidence for a definite diagnosis. The differential diagnosis between scurvy, congenital syphilis and rickets has been ably discussed by Wimberger and again by Bromer.

The latter author divided the course of the disease into four stages for purposes of roentgenologic classification.

Prominent Zone of Preparatory Calcification.—Since the broadening and intensity of the lime salt shadow at the zone of preparatory calcification is a measure of arrested length-growth of the scorbutic bone, during which time calcium continues to be deposited but, by virtue of the slowing or cessation of growth, fails to be distributed evenly on normal trabeculae and piles up at the osteochondral

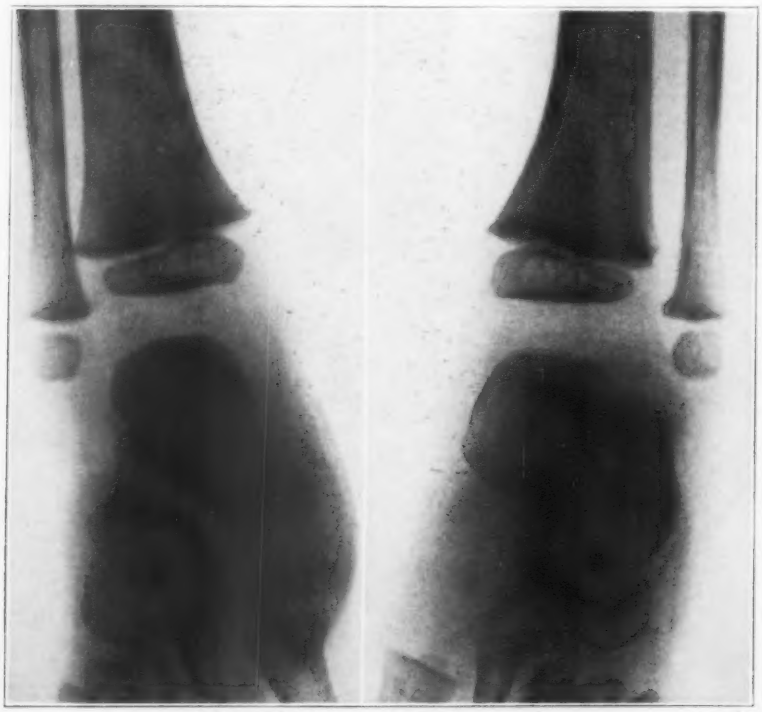

Fig. 13 (P. D.).—Condition twenty-three months after active scurvy, showing rarefied centers in the tibial centers of ossification. The fibular centers of ossification, which were not formed at the time of the active disease, are normal. Faint changes may be seen in the tarsal bones.

junction, it may be surmised that this sign will show up less conspicuously in early forms of the disease, before definite clinical symptoms have begun to appear or when they have been present for a short time only. This supposition is clearly borne out in selected cases of this description. Moreover, since the coincidence of active rickets with early scurvy delays lime salt deposit, one may expect at times to look in vain for this sign when such a clinical combination exists. Several observers (Wimberger, Pelkan and Bromer) have noted the fact that one often finds an increase in both the width and the intensity

of the zone of preparatory calcification during the first few days after treatment is begun (figs. 48, 49 and 50). In rachitic patients this is particularly apt to be true, but it appears too rapidly to be explained on the basis of healing rickets; however, we have seen such progressive increase continue for a period of six weeks during which the patient received intensive antirachitic as well as antiscorbutic treatment. Often during the early stages of healing the zone of preparatory calcification

Fig. 14 (P. D.).—Condition twenty-three months after active scurvy, showing rarefied areas in the centers of ossification of the femora and tibiae. It should be noted that the centers of ossification give evidence of growth on all sides except the one next the epiphyseal line. Transverse calcified lines in the shafts have disappeared. The metaphyses show Frank's "anvil" deformity.

becomes not only broader but more irregular in outline, and may take on a foamy appearance (figs. 39 and 42). Wimberger showed how in healing rickets the resumption of lime salt deposition at the zone of preparatory calcification may at times simulate in the x-ray the picture of acute scurvy. In healing scurvy, growth is restored at a normal or even an accelerated rate; and in its course the new zone of preparatory calcification travels away from its former position, with the following

results: at first the shadow broadens (in the direction of the long axis and away from the center of the bone), constituting the juxtaposition of a normal zone alongside the scorbutic zone of increased lime salt deposit; with further normal growth, this single broad line or zone splits into two—a normal zone of preparatory calcification at the osteochondral junction, and the residual band of lime salts due to the scorbutic process, now being buried, as it were, in the shaft of the bone and showing a reluctance to be absorbed—which are separated by an

Fig. 15 (P. D.).—Condition twenty-three months after active scurvy. Lateral view of right knee, showing disappearance of evidence of posterior separation of the femoral epiphysis.

ever widening strip of normal trabecular bone, or, when there is accompanying rickets, the homogeneous type of trabecular bone which is peculiar to healing rickets (figs. 5, 26, 27, 28, 30, 31, 32, 45, 46, 47, 48, 49 and 50). During the course of the ensuing weeks or months, the scorbutic scar remains as a transverse line of calcification, becoming in time gradually less conspicuous until its final removal by osteoclasis, but marking, so long as it remains, the date of the disease process from which subsequent growth may be measured (figs. 6 and 53). Just as the manifestations of this sign—the whole metamorphosis through

which the scorbutic epiphyseal line passes—vary in intensity with the changes which the bone experiences in rate of growth, so the degree of its prominence varies not so much with the severity of the local clinical signs as with the rate of normal growth of bone at individual epiphyses. Thus, the sign is found, if it is to be found at all, at the lower end of the femur, at both ends of the tibia and fibula, at the upper end of the humerus and the lower ends of the radius and ulna, and rarely if ever at the lower end of the humerus and the upper end

Fig. 16 (P. D.).—Condition twenty-six months after active scurvy.

of the ulna. Growth of the ribs takes place rapidly, but owing to difficulties of roentgenographic technic, these changes are rarely distinguishable in the ordinary x-ray of the thorax. The formation of transverse lines of calcification in the shaft of long bones in conditions other than scurvy has been described in detail by Eliot, Souther and Park.[10] In one of our own patients, transverse lines caused by scurvy

10. Eliot, Martha M.; Souther, Susan P., and Park, E. A.: Transverse Lines in X-Ray Plates of the Long Bones of Children, Bull. Johns Hopkins Hosp. **41:** 364 (Dec.) 1927.

were plainly visible 117 weeks after the beginning of treatment for the disease in its acute stage, but the majority of them disappear more quickly. In general, the length of time during which they persist depends partly on the severity of the original disease process and partly on the age of the patient and the rate of growth of the bone. They disappear earlier in young patients.

Fig. 17.—E. B., age on admission, 15 months. One month before admission, left leg had been put in plaster cast for supposed anterior poliomyelitis; lately, right leg tender. Typical scorbutic attitude of left leg; tenderness of both lower extremities, with swelling at lower part of left thigh and left leg; hemorrhages around teeth; miliaria; temperature, 102.4 F.; microscopic hematuria. The roentgenogram, taken one week after admission, shows rarefaction of all bones, with blurring of trabecular markings and thinning of cortices; sharp outlines of centers of ossification; dense zone of preparatory calcification; subperiosteal fractures in both tibiae; spurs on both femora, and early deposit of lime salt in elevated periosteum at the lower end of the left femur.

Spur at Lateral Edge of Epiphyses.—An increase in width of the zone of preparatory calcification at right angles to the long axis of bone gives rise to the formation of spurs, which in their

typical form jut out as prolongations of the epiphyseal line beyond the limit of the normal cortical shadow at that level (figs. 1, 3, 7, 17 and others). The end of the bone may look like a metal rod, the end of which has been hammered down and caused to mushroom out. They occur under three different conditions: (1) in association with lateral separation of the epiphysis, when the whole epiphyseal line shifts sideward with respect to the rest of the shaft, the

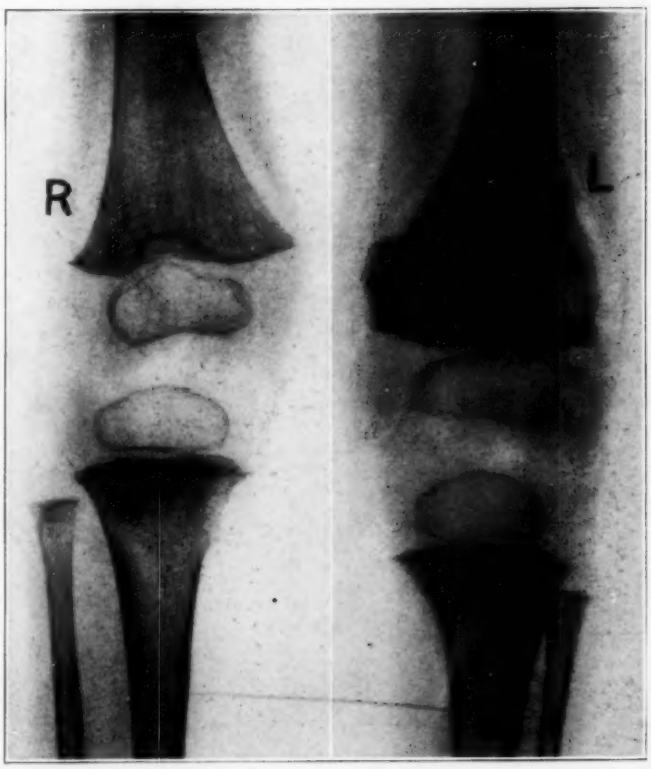

Fig. 18 (E. B.).—Condition four weeks after admission, showing increase in density of all bone shadows with reappearance of trabecular markings and dense shadow of elevated periosteum, with lamellated structure. The fracture deformity persists in the right tibia. In this case the zone of preparatory calcification is not so conspicuous as frequently occurs, and the adjacent zone of rarefaction is indistinguishable.

line of cleavage occurring in the zone of rarefaction; (2) as an accompaniment of the healing process in the presence of a subperiosteal hemorrhage at the epiphyseal end of the bone, the earliest deposition of lime salts in the elevated periosteum often becoming evident first in that portion of it which is immediately adjacent to the epiphyseal

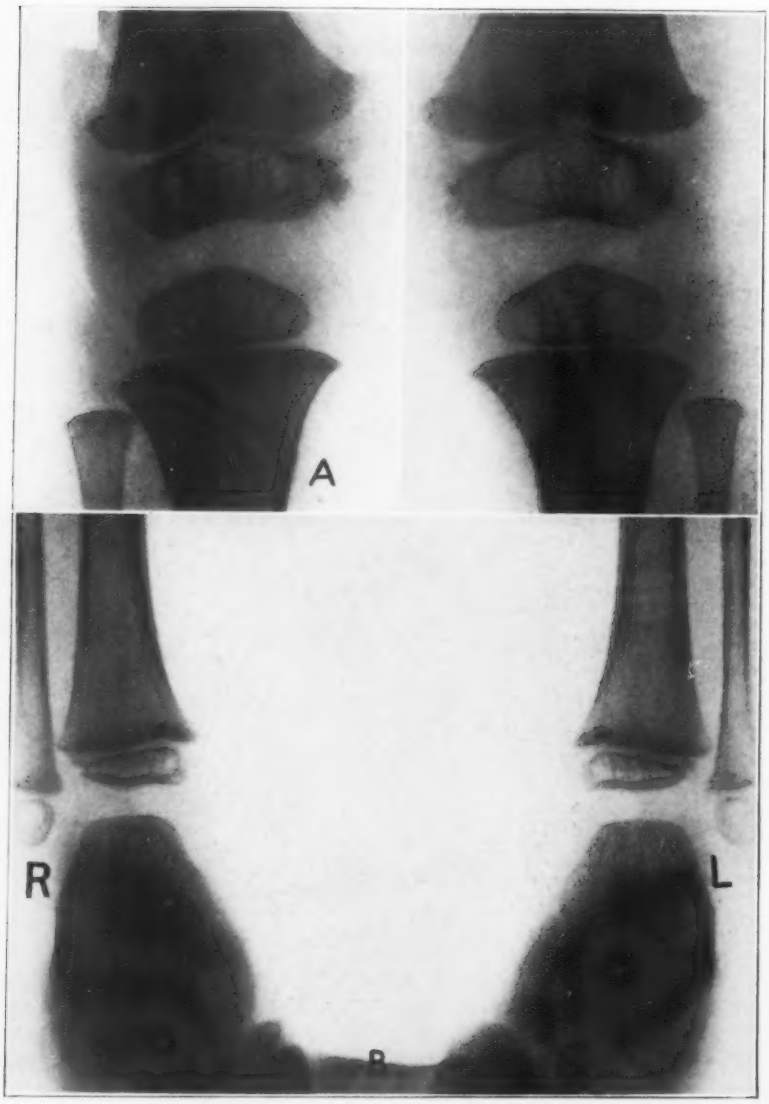

Fig. 19 (E. B.).—*A*, condition fourteen months after active scurvy, showing scars of scorbutic process in centers of ossification. The fraying of the femoral centers is not attributable to scurvy. In the right tibia several transverse calcified lines may be distinguished, but it is impossible to say which of these, or whether any of them, is attributable to the active scurvy. *B* is a detail from the same film. The changes persisting in the tarsal bones should be noted. In this patient, the lower center of ossification of the fibula was formed at the time of the active disease process, and therefore shows central rarefaction.

line, to which, in fact, the periosteum remains firmly attached even when it is stripped away from the entire circumference of the shaft, and (3) more rarely in the absence of either demonstrable epiphyseal separation or subperiosteal hemorrhage. Combinations of the first and second types commonly occur. The recognition of spurs is not always

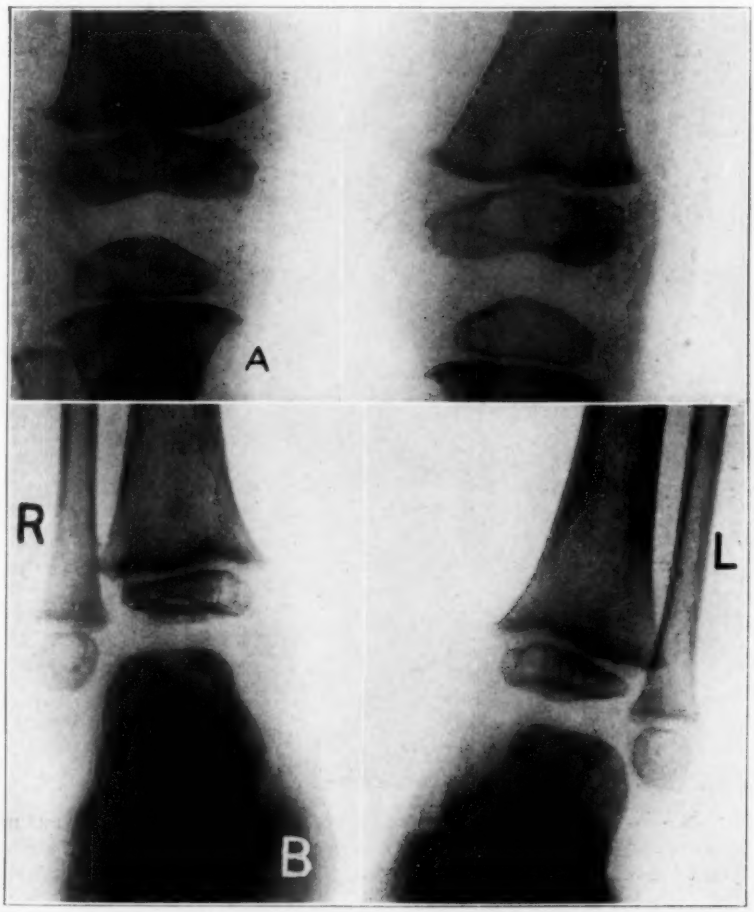

Fig. 20 (E. B.).—*A*, condition seventeen months after active scurvy. *B*, detail from same film.

easy. In the first place, when rickets has produced cupping of the ends of the long bones as it does at those epiphyses where the rate of growth is normally rapid, not only are the limits of the zone of preparatory calcification itself more difficult to make out on account of the accompanying poverty of lime salt deposit but the rachitic process itself may lead to an increase in width of the metaphysis so that the zone of

Fig. 21.—M. F., age on admission, 7 months. Tenderness when disturbed (duration not given). Small, anemic, dehydrated. Typical scorbutic attitude; swelling of upper third of both upper arms, upper part of both thighs, and lower part of both legs; temperature, 101.8 F. Serum calcium, 10 mg. in 100 cc.; phosphorus, 3.5 mg. in 100 cc. The roentgenogram, taken one day after admission, shows marked diminution in density of the bone shadows, with blurring of the trabeculae and thinning of the cortices. The centers of ossification show conspicuous scalloped borders. The shadows of the lower ends of the femora and tibiae show that the zone of preparatory calcification is a surface, not a line. Zones of rarefaction are well marked in the upper ends of the tibiae. Spur formation and multiple epiphyseal separation, including the upper ends of both femora, are preserved. There is a faint outline of the periosteal shadow around the femora. The separation of the right tibia and fibula by a hematoma should be noted. The tarsal bones show changes similar to those in the centers of ossification.

preparatory calcification projects out beyond the normal diaphyseal thickness at that point. The distinguishing feature under these conditions, however, is the direction in which the prolongations of the epiphyseal line point; for such rachitic spurs are directed at least to

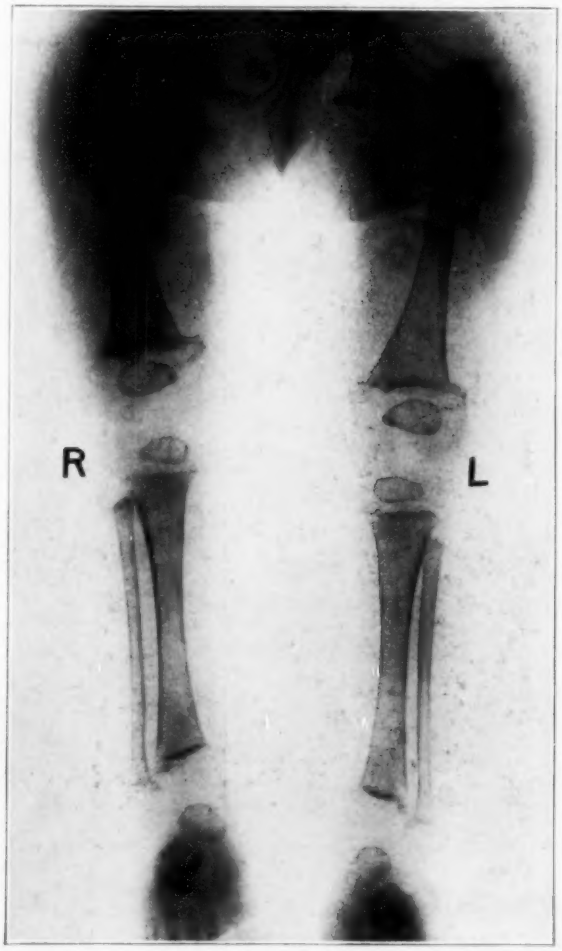

Fig. 22 (M. F.).—Condition five days after admission, showing increase in density. The periosteal shadows in the thighs are more definite. The reapproximation of the right tibia and fibula should be noted.

some extent toward the joint, whereas scorbutic spurs either project straight out at right angles to the long axis of the bone or else tend to curve back toward the middle of the shaft. However, there are exceptions even to this general statement; for example, in locations where the normal epiphysis shows a certain degree of concavity toward the

joint, as in lateral views of the lower end of the femur, typical scorbutic spurs may jut out in a direction which simply continues the curve (figs. 9 and 23). In one of our most severe cases there was an enormous hematoma at the lower end of one femur in which the blood was under such tension that it ballooned the periosteum downward in the direction of the knee joint; when calcification of this elevated periosteum first began to be visible in that portion of it which was immediately adjacent

Figure 23

Figure 24

Fig. 23 (M. F.).—Lateral view of right leg, taken on same date as figure 22, showing anterior separation of the femoral epiphysis, giving rise to a long spur.

Fig. 24 (M. F.).—Lateral view of left leg, taken on same date as figure 22.

to the zone of preparatory calcification, it gave rise to the appearance of spurs projecting in the direction of the joint as well as away from the shaft.

Another source of difficulty in the interpretation of spurs arises from the fact that the zone of preparatory calcification actually represents in the x-ray the projected shadow of a disk, and not even always a plane disk, as large as the cross-section of the metaphysis at that level; and when the

rays traverse it at an angle, the resulting impression on the film may be not a sharply defined line but a blurred ellipse or figure-of-8 or an even more intricate shape (figs. 21 and 22). Consequently, often the most that can be said in interpreting such a film is that there is some extension outward from the expected margin of the zone of preparatory calcification, the nature of which cannot be more precisely defined. To a certain degree spur formation may be taken as an index of the severity of the individual case, for in severe forms of scurvy it tends

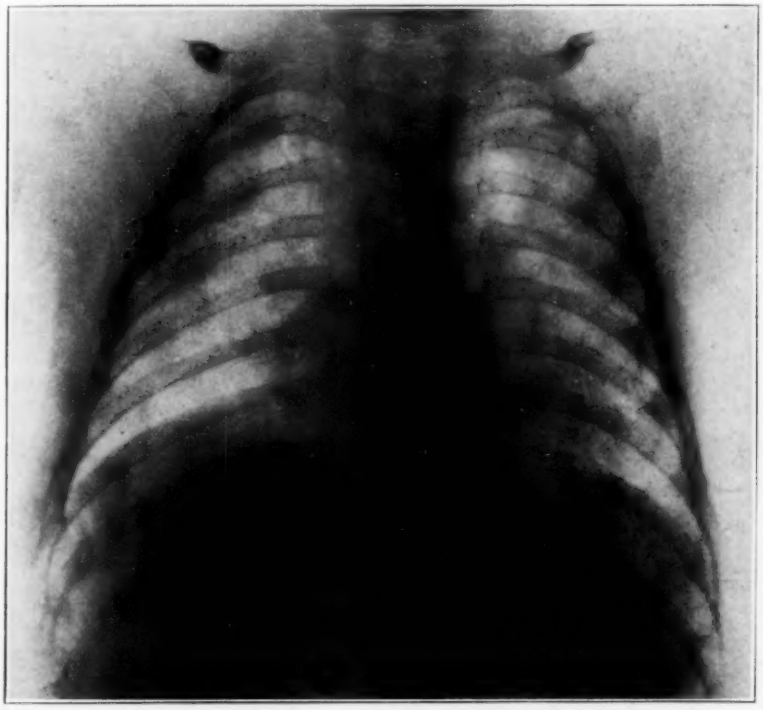

Fig. 25 (M. F.).—Multiple epiphyseal separations at vertebral ends of ribs, on same date as figure 22.

to be most marked in those regions in which other manifestations of the disease process are most marked, and in mild forms it may not be present at all. We have independently confirmed Bromer's observation that spurs may occur in situations in which serial examinations over a period of weeks have failed to demonstrate the presence either of separation of the epiphysis or of a recognizable subperiosteal hemorrhage.

Zone of Rarefaction.—The zone of rarefaction in the diaphyseal shadow immediately adjacent to the zone of preparatory calcification,

in the direction of the center of the bone, has been called by Bromer the framework marrow zone, since it appears to be caused by the replacement of normally calcified trabeculae in that part of the marrow cavity by the fibrous marrow peculiar to scurvy (figs. 1, 2, 4, 7, 10 and

Fig. 26 (M. F.).—Condition two weeks after admission, showing increase in density, with diminution in size, of the periosteal shadows.

others). It was first described by Lehndorff.[11] Pelkan gave it the name of scurvy line, since he did not find it in any other condition. An appearance at times closely resembling it is found at a much earlier age, for example, in infants with syphilitic osteochronditis in the first

11. Lehndorff, H.: Arch. f. Kinderh. **38**:161, 1904.

few months of life. This picture has been admirably described by Wimberger, Alexander [12] and Fraenkel,[13] and since age incidence alone prevents any serious difficulty in differentiation of the two processes, we shall not give further details of the roentgenographic distinction. The scorbutic zone of rarefaction depends for its formation on a regional failure of lime salt deposit; and Wimberger has pointed out that when rarefaction of the entire bony shadow is marked and trabeculae are

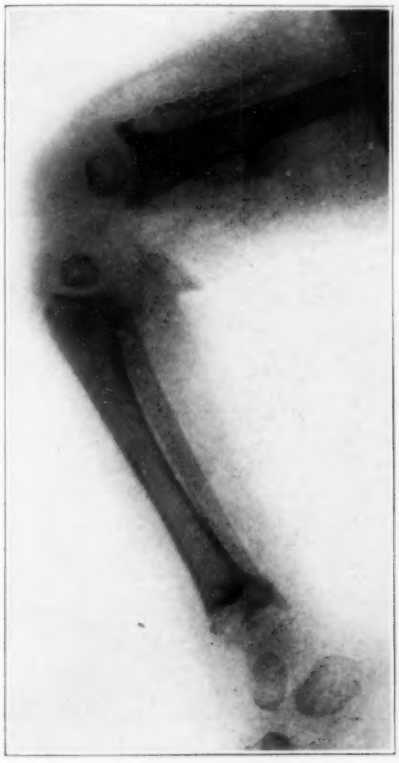

Figure 27

Figure 28

Fig. 27 (M. F.).—Same date as figure 26. Lateral view of right leg.
Fig. 28 (M. F.).—Lateral view of left leg, taken on same date as figure 26.

no longer distinguishable in any portion of the marrow cavity, it may then be impossible to demonstrate any further degree of rarefaction in the scurvy line. However, it may be impossible to detect even in cases in which the generalized process of rarefaction has not been sufficiently

12. Alexander, Béla: Die ostealen Veränderungen bei kongenitaler Syphilis, Leipzig, J. A. Barth, 1915.

13. Fraenkel, E.: Die kongenitale Knochensyphilis im Röntgenbilde, Hamburg, Lucas Gräfe & Sillem, 1911.

severe to obliterate all trabecular markings. It is absent in many cases, especially early ones, even though the zone of preparatory calcification is prominent; but, on the other hand, in two of our cases the zone of rarefaction was present when excessive calcification of the epiphyseal line could not be claimed. During the healing process the zone of preparatory calcification broadens still further and becomes more conspicuous, as has already been shown, and at the same time calcification of the existing trabeculae in the marrow cavity occurs, as will be described later in the paper, so that for a period of from one to four

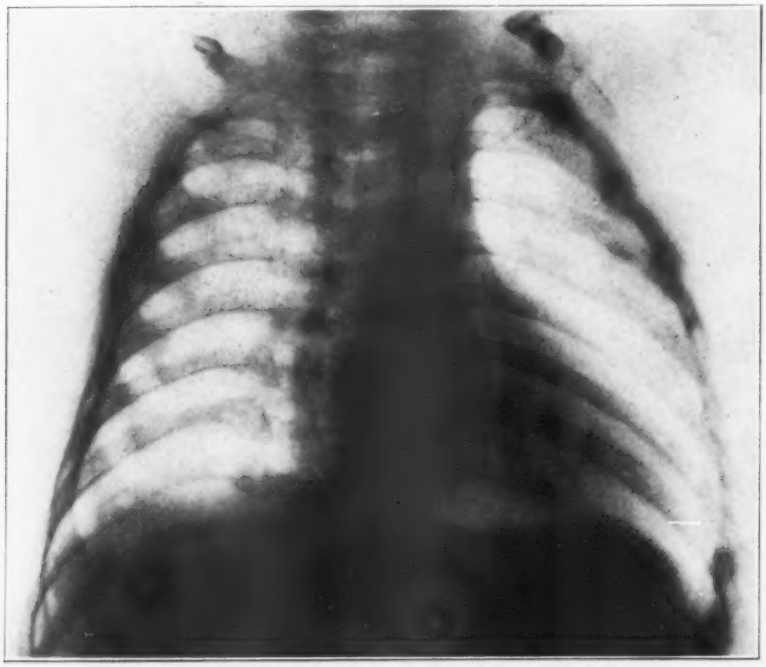

Fig. 29 (M. F.).—Condition three weeks after admission, showing shadow of calcified elevated periosteum at vertebral ends of several ribs and scorbutic lesions in humeri.

weeks from the commencement of treatment the zone of rarefaction, which contains few if any trabeculae and consequently does not share at once in the calcifying process, becomes more and more conspicuous (figs. 1, 2, 7, 10, 36, 39, 42 and 45). At the end of this time, however, trabeculae commence to invade it from the adjacent spongiosa, and it gradually becomes indistinguishable.

Dense Margin of Epiphyseal Center of Ossification and Rarefaction of Central Portion.—The growth process in the centers of ossification is analogous to that at the ends of the long bones, with the modification

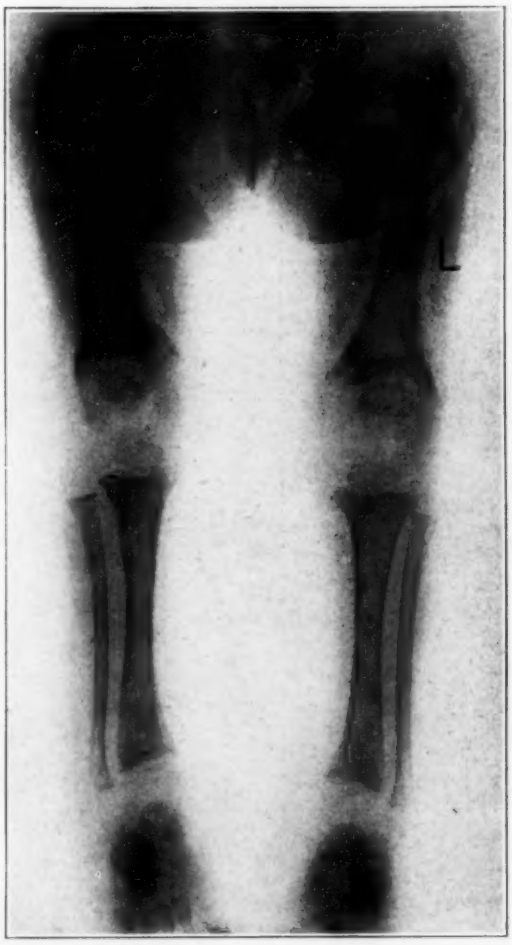

Fig. 30 (M. F.).—Condition six weeks after admission. Comparison with figure 22 and figure 26 shows the rapidity of resorption of subperiosteal hemorrhages of femora with contraction of periosteal shadows. The tibiae and fibulae show a typical picture of healing rickets most marked in the proximal end of the fibulae and the distal ends of the tibiae. The bowing of the fibulae is probably a rachitic phenomenon. Trabecular markings have reappeared, particularly in the older portions of the bones.

that it proceeds from a surface roughly spherical in form instead of from one roughly plane; that it is, in general, slower in rate and, finally, that its rate is not uniform in all parts of the surface, being most rapid on the side toward the joint and on the lateral aspects; it is slower—in fact, it is almost imperceptible in centers more than 1 or 2 cm. in diameter—on the side toward the diaphysis. The changes

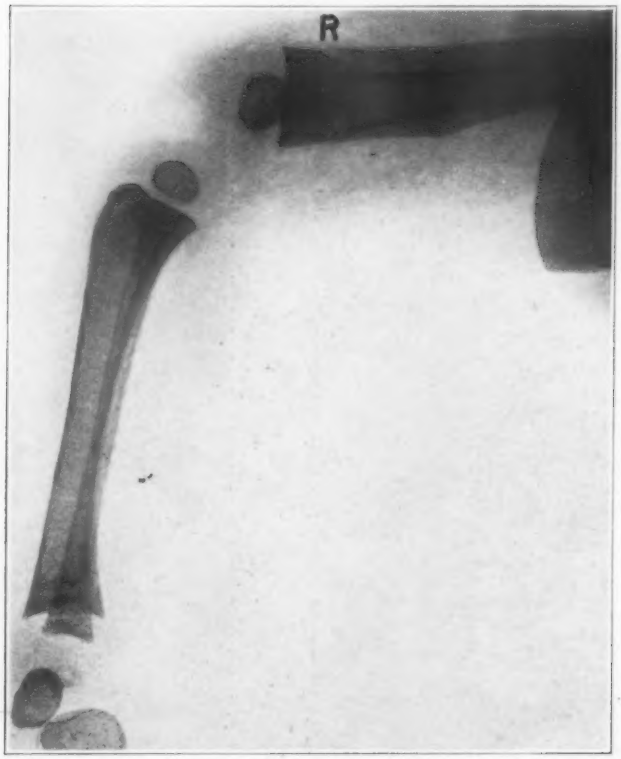

Figure 31

Figure 32

Fig. 31 (M. F.).—Lateral view of right leg, taken on same date as figure 30.
Fig. 32 (M. F.).—Lateral view of left leg, taken on same date as figure 30.

which have been described in the breadth and density of the zone of preparatory calcification, with its adjacent zone of rarefaction and beyond that a region of blurred, hazy trabecular marking, have their counterpart in the centers of ossification (figs. 1, 2, 3, 4 and others). The line of opacity marking the periphery becomes broader and more opaque in scurvy, except on the side next to the diaphysis, where growth is slow; often the central part, on the other hand, increases

in transparency and loses its customarily sharp trabecular markings. If these are not completely absent, they at any rate become faint and often take on a delicate foamy, soap bubble-like appearance. These roentgenologic signs of scurvy were first described by Reyher [14] and

Fig. 33 (M. F.).—Condition twenty months after active scurvy, showing residual markings in the centers of ossification; slightly exaggerated bowing of the left femur, and absence of transverse calcified lines.

later by Gött,[15] but Wimberger was the first to insist on their specificity. According to the last named author, they are not found in any

14. Reyher, P.: Das Roentgenverfahren in der Kinderheilkunde, Berlin, 1912.

15. Gött, H.: Roentgenuntersuchungen in der Kinderheilkunde, in Rieder-Rosenthal; Lehrbuch d. Roentgenkunde, Berlin, 1918.

other condition. We must take exception to this statement, since we have seen an almost identical picture during the healing process in an unusually severe case of rickets in a colored child, aged 17 months, following intensive treatment with cod liver oil and the ultraviolet ray lamp, in which the dense shadow of new bone formed at the periphery

Fig. 34 (M. F.).—Lateral view of right leg, taken on same date as figure 33, showing exaggerated anterior bowing of the femur, with thickening of the cortex on the concave side.

of the centers of ossification contrasted sharply with the rarefied interior. Under such circumstances, one must take the clinical course of the patient into consideration in order to avoid making a false diagnosis of scurvy. Simple atrophy may cause a rarefaction of the entire

shadow of the centers of ossification, but it is not associated with a concentration of the lime salt deposit at the periphery.

These changes in the centers of ossification mark a temporary slowing or cessation of growth during the active period of scurvy, just as is the case in their analogues at the epiphyseal ends of the long bones. In

Fig. 35 (M. F.).—Lateral view of left leg, taken on same date as figure 33, showing thickening of cortex on concave side.

the healing process, again, a similar train of events ensues. Growth is resumed in those portions of the periphery in which the rate of growth is normally appreciable, and the annular shadow becomes broader and more conspicuous in its lateral aspects and on the side next to the joint. At the same time, trabeculae reappear in the center; or the foamy, faint

Fig. 36.—C. R., age on admission, 1 year. Swelling of right ankle, one week; of both elbows and wrists, one day. Marked generalized tenderness, with all extremities held in flexion; swelling of lower part of right leg, with ecchymosis, bony crepitus and false motion; crepitus in left shoulder; hemorrhages in gums; miliaria; bronchopneumonia. Serum calcium, 10.4 mg. in 100 cc.; phosphorus, 1 mg. in 100 cc. The roentgenogram, taken two days after admission, shows rarefaction of all bones; blurring of the trabeculae and thinning of the cortices; conspicuous periphery of the centers of ossification, with central rarefaction; prominent zones of preparatory calcification; spurs; lateral separation of lower epiphysis of the right tibia, with wedging apart of the tibia and fibula; sharp outline of the tarsal bones and subperiosteal fracture at the upper end of the left tibia.

shadow of altered trabeculae increases in density and definition (figs. 3, 4, 5, 6 and others). On account of the fact that even in a normal center of ossification the margin is often slightly accentuated, there may be produced at some stage of the healing process the appearance of a double ring—the outer one the healthy growing margin, the inner one the unabsorbed shell of lime salts laid down during the active phase of the illness. Wimberger described this picture in some detail, but in our

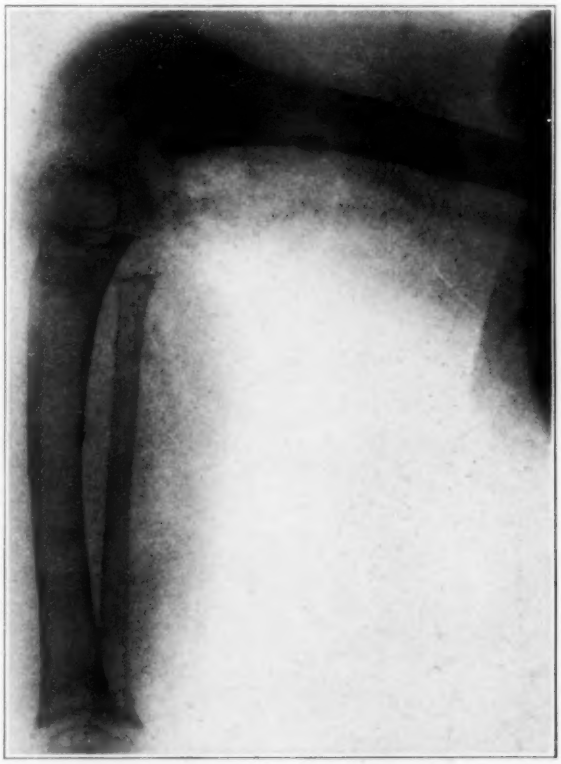

Fig. 37 (C. R.).—Lateral view of right leg, taken on same date as figure 36.

experience it is more common to find that the outline of the center of ossification keeps on broadening peripherally, at varying rates in different portions, as has already been outlined, burying the scorbutic scar deeper and deeper in the normal fine grained spongy bone of more mature centers of ossification. Probably owing to the handicap of impaired circulation the central nucleus, which at the time of the active disease represented the entire center of ossification at that moment, tends only with extreme slowness to resume its normal appearance (figs. 13 to 16, 19, 20, 33 to 35 and 51 to 55). It retains its essentially rarefied aspect,

in contrast to the framework of bone or more recent formation which surrounds it; the intensified periphery and the soap bubble trabecular structure, far more coarse than that of the surrounding bone, may persist for years in the severe cases of long duration. We have observed this picture in a boy, aged 12½ years (fig. 55), who had scurvy when he was 10 months old, and in another boy, aged 13, in whom scurvy occurred at 8 months. This is several years longer than other

Fig. 38 (C. R.).—Lateral view of left leg, taken on same date as figure 36.

examples reported (Wimberger, "four or five years"; Schwartz, four years, three months; Bromer, five years); in fact we have found this sign persisting in at least one epiphysis in all but one of our old cases which we have been able to follow. Although the absence in uniformity of the tube distance and the direction of the ray prevents a strictly quantitative treatment of the subject, superposition of the original films on the later ones shows at least that for a few years the outline of the rarefied area remains essentially the same and marks the size of the center of ossification at the time of the active disease, though in the

course of the later years it shrinks perceptibly. If a center had not been formed at the time the patient suffered from scurvy, its subsequent development will have been entirely normal, at least as far as this scorbutic change is concerned, for we have not seen a patient who showed two separate periods of active scurvy. Thus, with some knowledge of

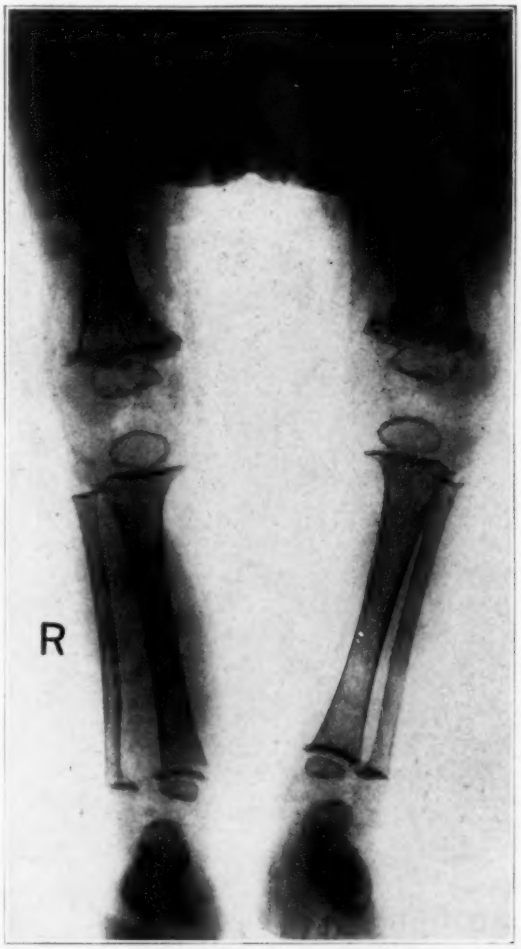

Fig. 39 (C. R.).—Condition one week after admission, showing increase in density, with reappearance of trabecular markings and calcification of elevated periosteum at the lower end of the right fibula.

the age at which the various centers of ossification appear in the infant's skeleton and of their normal rate of growth, although both of these vary rather widely within normal limits, one may, from examination of the older child's x-ray photographs alone, make an approximate estimate of the age at which active scurvy occurred.

It is interesting that this late manifestation, the description of which was first published by Wimberger from his Vienna clinic, was independently observed at about the same time in Philadelphia, Milwaukee and New York. Examination of Fraenkel's photographs,[16] moreover, discloses a clear example of it in the head of the femur of one of his patients, of whom exposures taken shortly after the acute stage of the disease and again more than three years later are reproduced. It is not claimed by us that a roentgenologic demonstration of a rarefied central

Figure 40

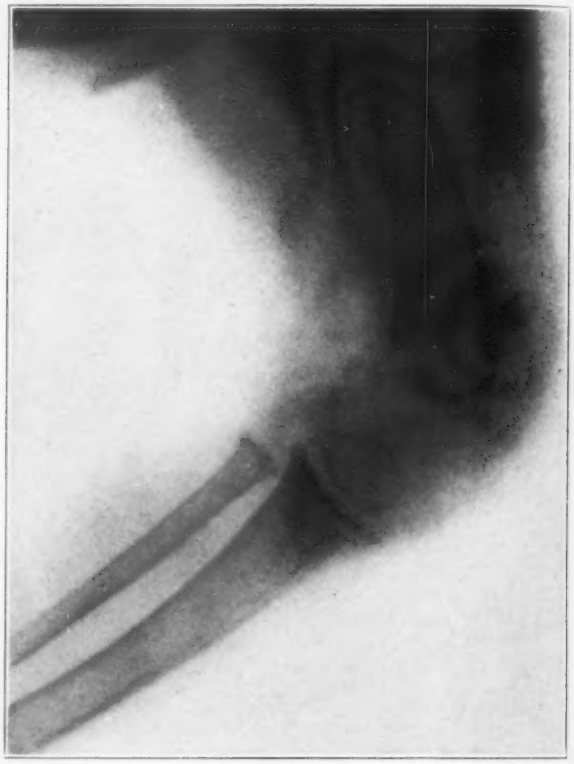

Figure 41

Fig. 40 (C. R.).—Lateral view of right leg, taken on same date as figure 39.

Fig. 41 (C. R.).—Lateral view of left leg, taken on same date as figure 39. The lower center of ossification of the femur does not show exaggeration of its outline in that portion of it adjacent to the epiphysis.

nucleus affords certain evidence of previous scurvy, since a temporary arrest of growth with rarefaction at the center of the centers of ossification is the mechanism of this scorbutic bone change, and there is no theoretical reason why this could not occasionally result from some

16. Footnote 8, tables II and IV.

other cause. We have observed somewhat similar "scars" in a patient with acute lymphatic leukemia as well as in a patient with anemia of "von Jaksch's" type (fig. 56).

The small bones of the wrist and foot have an anatomic structure somewhat comparable to that of the centers of ossification of the long

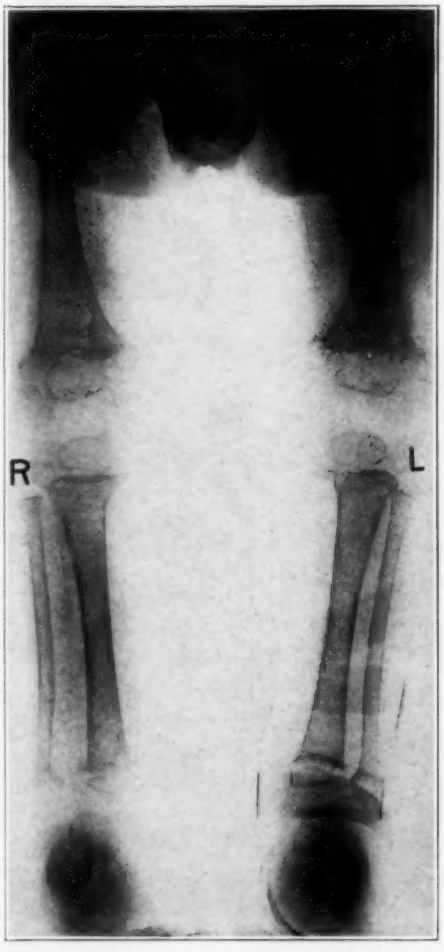

Fig. 42 (C. R.).—Condition two and a half weeks after admission. The zones of preparatory calcification at the lower ends of the femora show a foamy appearance. The outlines of the elevated periosteum begin to be visible.

bones, and in marked cases of scurvy they share the same changes as the latter, including the later manifestations just described. However, owing possibly to the fact that they have a richer circulation or at least a freer communication with the body fluids on which calcification and

growth of bone depend, the changes at their centers tend to disappear long before the mark of the disease has left the centers of ossification. We have observed cases in which residual effects were noticeable for several months after the clinical cure of scorbutic symptoms, but none so far comparable to the instance recorded by Schwartz four and a quarter years after discharge from the hospital. His photographs, indeed, offer a remarkable picture.

Figure 43

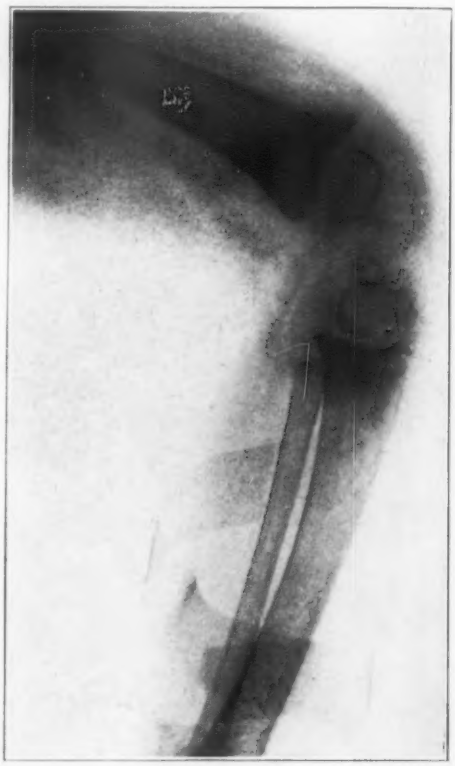

Figure 44

Fig. 43 (C. R.).—Lateral view of right leg, taken on same date as figure 42, showing "development" of coarse trabecular markings.

Fig. 44 (C. R.).—Lateral view of left leg, taken on same date as figure 42.

Separation of the Epiphyses.—Any discussion of separation of the epiphyses as it occurs in scurvy must be prefaced by a definition of terms. In the first place, when this time-honored expression is used in describing this severer manifestation of certain examples of the disease, it is essentially misleading. Not only the epiphysis is separated but the distal part of the diaphysis as well, carrying with it the zone of preparatory calcification, and the condition in reality represents a particular kind

of fracture. Wimberger suggested adoption of the term "separation of the metaphysis" as being more accurately expressive of anatomic considerations. In scurvy, the line of cleavage is through bone; in traumatic separation of the epiphysis, when scurvy is not implicated, the

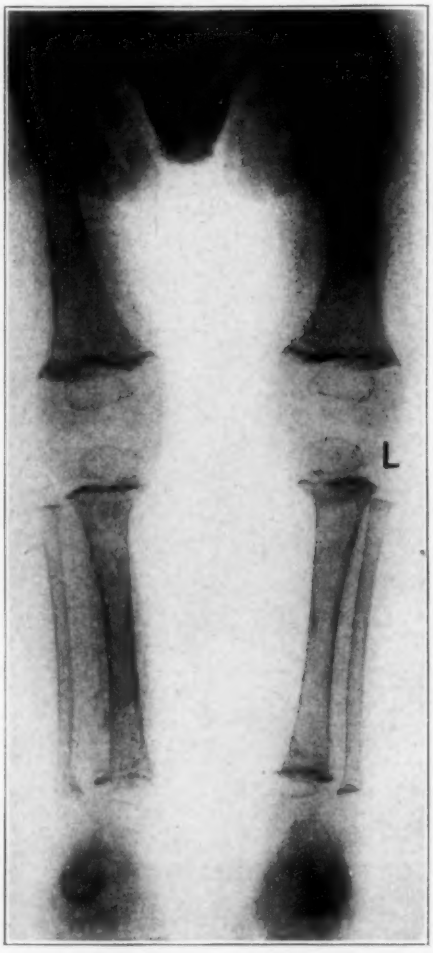

Fig. 45 (C. R.).—Condition four weeks after admission. Zones of rarefaction are now evident. The irregularities of the shadow of the elevated periosteum in the left femur and the right tibia should be noted. An increase in prominence of the zones of preparatory calcification is present.

line of cleavage is through the epiphyseal cartilage; and the two differ so clearly in their causation, pathologic anatomy, treatment and prognosis that the distinction, which, to be sure, is not difficult, must be kept in mind. We shall keep the term "separation of epiphyses," but shall discuss only the scorbutic form.

The frequency with which one makes the roentgenographic diagnosis of this complication depends entirely on the criteria which one adopts for determining the separation. When the entire epiphyseal fragment and zone of preparatory calcification are displaced laterally or anteroposteriorly so that the center of ossification no longer lies in the long axis of the bone, there is no question about what it shall be termed. But there are other possibilities, and they occur with considerably greater frequence: the epiphyseal fragment may be jammed against

Figure 46

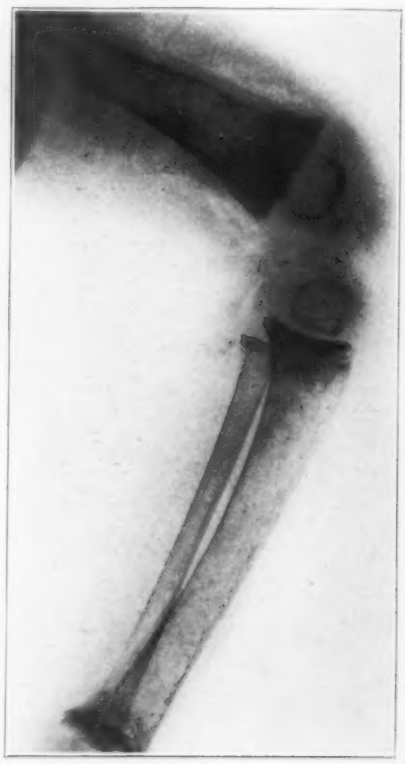

Figure 47

Fig. 46 (C. R.).—Lateral view of right leg, taken on same date as figure 45.
Fig. 47 (C. R.).—Lateral view of left leg, taken on same date as figure 45.

the diaphysis so that there is shortening, alteration of contour and obliteration of the zone of rarefaction; or, more commonly still, one portion of the epiphysis may be separated, giving rise to a widening of the zone of rarefaction at that point, while the rest of it maintains its customary relationship to the diaphysis. We are of the opinion that in both of these contingencies one should properly make the diagnosis of epiphyseal separation; and if this is done, the percentage of such lesions

in a given case or series of cases will be appreciably augmented. In one of our patients (C. R.), we counted nine such separations out of the eighteen possibilities comprised in a study of the upper ends of the humeri, the lower ends of the radii and ulnae and both ends of the femora, tibiae and fibulae. (Our films of the upper extremities have

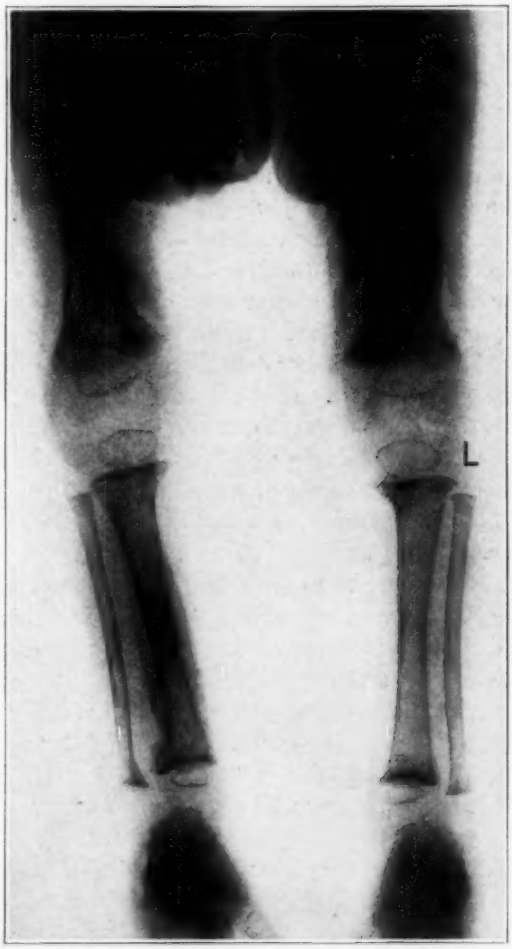

Fig. 48 (C. R.).—Condition five and a half weeks after admission, showing diminution in size, with increase in density, of the periosteal shadows.

not been included in the reproductions published herewith.) In two of our cases, epiphyseal separation, clearly distinguishable as lateral displacement of the zone of preparatory calcification with respect to the diaphysis, was visible at the spinal ends of several of the ribs (figs. 25 and 29). It is not uncommon for the number of separations to increase

during the first few days in the hospital, not because the handling of the patients is clumsy but because the bones are not in a condition to bear any type of handling, however gentle. Expert nursing will minimize the number of epiphyseal separations. From a clinical point of view, it is not necessary to protect such lesions by splints or other immobilizing devices except in the most marked instances, for within the first few days of treatment further displacement is promptly arrested, and the bone heals quite as well as if there had not been such a complication. As has already been described, distinct spur formation is often associated with separation of the epiphyses.

With the commencement of healing, the displaced fragment soon becomes fused into place in its new situation. The normal growth process is resumed both at the epiphysis and in the center of ossification and is unaltered except for possible stimulation by the change of position. If there has been impaction, with obliteration of the zone of rarefaction, the changes in the latter that have already been described will not be discernible; there will merely be an increase in density of the entire bone shadow, with better definition of the trabecular markings. On the other hand, when the zone of rarefaction has been widened by a separation away from the diaphysis, a less frequent occurrence, there may be some delay in the bridging of this gap with new trabeculae. Lateral separation of any degree of severity is invariably accompanied by the simultaneous occurrence of a hemorrhage beneath the adjacent periosteum, in which the first appearance of calcification shows clearly the attachment of periosteum to the margin of the zone of preparatory calcification, persisting even when it is stripped free from the entire length of the diaphysis (figs. 1, 23 and 45). The angle between the cortex of the diaphysis and the projecting shelf of the displaced zone of preparatory calcification does not contain a lime salt shadow in the early roentgenograms taken before calcification of the elevated periosteum has become distinguishable, but eventually fills with the dense shadow of calcified periosteum and hematoma. The changes which the latter undergoes will be more fully considered later. But one may point out here that the displaced epiphysis never returns to its former position, but slowly causes the diaphysis to create for it a new medullary canal by reinforcement of the cortex on one side and erosion on the other, so that eventually the center of ossification, often by this time considerably enlarged and altered in shape through the natural evolution it undergoes in normal growth, once more finds itself in line with the central axis of the shaft.

Ground-Glass Appearance of Medulla of Long Bones.—The ground-glass appearance of the medulla of the long bones, with clouding of the trabecular structure, has been found by us to be less constant in fresh cases of scurvy than certain authors, especially Pelkan, have

found it. We were able to identify it in about one fifth of our cases during the first week of treatment. A strict interpretation of this sign presupposes a uniformity and perfection of roentgenographic technic which we have not been able to command; if the patient has moved slightly during the exposure or if the film has been overexposed, the reading is attended with difficulties, so that it is not at all uncommon to find this appearance in the bones of nonscorbutic infants. However,

Figure 49

Figure 50

Fig. 49 (C. R.).—Lateral view of right leg, taken on same date as figure 48.
Fig. 50 (C. R.).—Lateral view of left leg, taken on same date as figure 48.

a certain degree of rarefaction of the entire bone structure is a practically constant accompaniment of acute scurvy, and serial roentgenograms made during the first three weeks of treatment of a patient will often show a progressive clarification and increased definition of the trabecular structure which in retrospect shows plainly enough that the condition was an actuality and not artefact. Diffuse rarefaction of bone is not limited, of course, to scurvy but occurs also in rickets, in paralyses of

various sorts, and in still other conditions. The relative coarseness of the trabecular structure in rickets has been emphasized by Wimberger as an important differential point, in addition to characteristic changes at the epiphyses. In atrophy of disuse, particularly when it is of long standing, the long bones exhibit a relatively normal growth in length but a distinctly subnormal growth in thickness. That the rarefaction found in scurvy is not simply a manifestation of atrophy of disuse caused by the coincident tenderness of the acute phase of the disease is attested by its occurrence in patients whose history of tenderness and loss of function dates back only a day or two, and in parts of the body in which tenderness is not discernible.

After treatment has been begun, increase in density of the whole bone shadow and progressive definition of the trabecular markings, which often develop out like the image on a photographic plate, continue rather rapidly for from two to four weeks until they reach the normal degree, remaining normal from then on during the period of observation. The presence of rickets retards this process noticeably, even when the patient is being treated with cod liver oil. We are not in a position to state what the change would be in patients who showed both rickets and scurvy and in whom treatment was directed solely toward the cure of scurvy.

Thinning of the Cortex and Hemorrhage into the Soft Parts.—Thinning of the cortex is another roentgenologic sign of scurvy or of atrophy accompanying scurvy which is hardly of sufficient constancy to admit of its discussion in the progress of healing as shown by the x-ray. Likewise, the appearance which we have interpreted as hemorrhage into the soft parts is not one about which we feel qualified to make unconditional statements. It is common enough, when a patient with a severe, acute form of scurvy is first examined under the x-ray, to find gross deformity of the soft parts by shadows of indefinite outline which, in view of the clinical evidence of large ecchymoses, one is tempted to look on as the shadow of a large extravasation of blood between muscle planes, such as has been described by Aschoff and Koch[17] from postmortem examination of adult scorbutic patients during the Great War; yet as healing sets in and the deposition of lime salts in the periosteum permits one to determine accurately the extent to which it has been displaced from the shaft by subperiosteal hemorrhage, it is usually discovered that the subperiosteal hemorrhage (as distinguished from what we have called hemorrhage into the soft parts) is of sufficient size to explain all the swelling found. We regard it as impossible, before calcification of

17. Aschoff, L., and Koch, W.: Veröffent. aus d. Geb. d. Kriegs u. Konstitutionspathol., Jena, 1919.

Fig. 51 (C. R.).—Condition twenty-one and a half months after acute scurvy, showing residual lesions in the centers of ossification, except in those of the fibula, which were not formed at the time of the active disease, and numerous sets of transverse calcified lines of origin subsequent to the commencement of healing. The site of the zone of preparatory calcification at the time the scurvy was active is probably marked out in the lower end of the right tibia by the sudden transition from spongy bone to medullary canal. The right tibia and fibula have resumed their normal relationship. Some of the tarsal bones show central areas of rarefaction with coarser trabeculae.

elevated periosteum has shown the full extent of subperiosteal hemorrhage, to state exactly the extent of a suspected hemorrhage into the soft parts.

Superiosteal Fractures.—The mechanical weakening of long bones resulting from these processes occasionally gives rise to fractures of the shaft (figs. 8, 9, 17, 18, 36 and 37) aside from the special form of fractures which we have discussed under separation of the epiphyses. Characteristically, these occur in the spongy bone at the end of the diaphysis; they are subperiosteal in type and are usually without a definite line of cleavage but show as collapse or buckling of the trabecular structure at that point. Sometimes they give rise to shortening of the bone; at other times, because of their location on only one side they cause merely a turning of the epiphyseal line with slight angulation. Their healing is not accompanied by callus formation; the bone merely fuses in its new position, and with resumption of growth at the epiphyseal end, the angulation is quickly straightened out. Permanent shortening has not been observed. We have seen the angular deformity persist for a period of eight months after the institution of antiscorbutic treatment.

THE CALCIFICATION OF SUBPERIOSTEAL HEMORRHAGES AND RATE OF RESTORATION TO NORMAL

Probably the most dramatic phase of healing scurvy, from the roentgenologic point of view, is the series of changes through which a subperiosteal hemorrhage passes, especially if it is a large one. Before the healing process has set in, it is often impossible to state definitely from examination of roentgenograms whether subperiosteal hemorrhage has occurred, although large hemorrhages cause a diffuse increase in width of the shadow of the soft parts, as already described, at first without any definite outline of lime salt deposit. It is now well agreed that neither the freshly detached periosteum nor the hematoma itself casts a dense shadow, and it is not until treatment has caused the osteoblastic layer of the periosteum to deposit lime salts that the exact extent of the lesion can be mapped out. Fraenkel was led to assert the contrary, since he found dense shadows in certain patients in whom he considered the hemorrhage to have occurred at so recent a date that sufficient time had not elapsed for formation of new bone to have occurred. The present day interpretation of such a picture would be that the patient had wittingly or unwittingly received enough antiscorbutic vitamin previously to initiate the healing process. The evidence for this view has been reviewed by Wimberger.

It would be not only interesting as a contribution to the study of the pathologic physiology of scurvy, but extremely useful for the interpretation of x-rays, if one could state with precision the length of the interval between the institution of antiscorbutic treatment and the first

appearance of definite lime salt shadow in elevated periosteum. The problem has not been solved by dietetic experiments on guinea-pigs, since in these animals the type of lesion found in man cannot be reproduced; and in the study of infants the interpretation of signs is complicated in the first place by the possibility that treatment has pre-

Fig. 52 (C. R.).—Residual bowing of left femur, on same date as figure 51.

viously been received and in the second place by the fact that the coincidence of active rickets with active scurvy noticeably delays the deposition of lime salts. In studies of individual cases from our series in which roentgenograms were made before healing of any perceptible degree had taken place, the shortest observed time during which antiscorbutic treatment was administered before the elevated periosteum cast a definite shadow due to bone formation in its new position was

one week. Certain patients, of course, do not show elevation of periosteum in the long bones at any stage of the disease. Such, for instance, was a boy, aged 8 months, in whom the diagnosis was based on the history of tenderness, the presence of typical lesions in the gums, the characteristic attitude of scurvy, markedly painful extremities and a rapid clinical cure under treatment. X-ray photographs of the long bones taken on the first, twelfth and nineteenth days of treatment and after six weeks showed rickets but no change in the periosteum. From evidence of this kind we believe that tenderness may occur in scurvy without the presence of a subperiosteal hemorrhage large enough to show in the roentgenogram; and such observations may throw some light on the clinical picture which has been described as the tenderness of acute rickets.

When calcification of the elevated periosteum commences to appear, it is sometimes first found in that portion of the periosteum which attaches to the zone of preparatory calcification. This functional peculiarity of this particular portion of an anatomically uniform structure like the periosteum may depend on local circulatory conditions, but may also have some other cause of a different nature. In the normal growth process of long bones, osteoclasis is especially active in this portion of the periosteum, since the width of the newest portion of the shaft, where it is first laid down at the zone of preparatory calcification, is greater than that of older portions farther toward the center of the shaft; and it may be that under such abnormal conditions as exist when the periosteum is stripped away from the shaft in scurvy, this activity is reversed and new bone is formed with particular rapidity. Since blood vessels enter the periosteum at several points and not especially at the level of the epiphyseal line, an explanation of this phenomenon based on circulatory anatomy is less plausible. After the first appearance of calcification, in the course of the next day or two a delicate shadow of lime salt deposit may be detected throughout the whole course of the elevated periosteum, definitely marking out the extent of the hematoma. Subsequently, two processes occur with considerable rapidity: an increase in the intensity of the shadow of new bone laid down by the periosteum, and a shrinkage of the hematoma. During the first few days during which the periosteal shadow is visible, there may be a lack of uniformity in its density, especially when it is associated with rickets, and it may show irregularities at its periphery (figs. 2 and 45); but parallel with the increase of intensity of the shadow a progressive homogeneity occurs and by the time five or six weeks have elapsed since the commencement of treatment, sufficient new bone has been formed to cast a uniform fusiform shadow which is often of sufficient density to obscure the enclosed cortical outline. The tendency of all such shadows is to contract down until finally the normal contour of the bone is restored, and this may take anywhere from a month to several months

(thirty-five weeks in our most protracted case), depending on the severity of the local lesion and also to a large extent on the degree of concomitant rickets. But in rare instances, even when there has been no separation of epiphyses, a local thickening of the cortex on the side

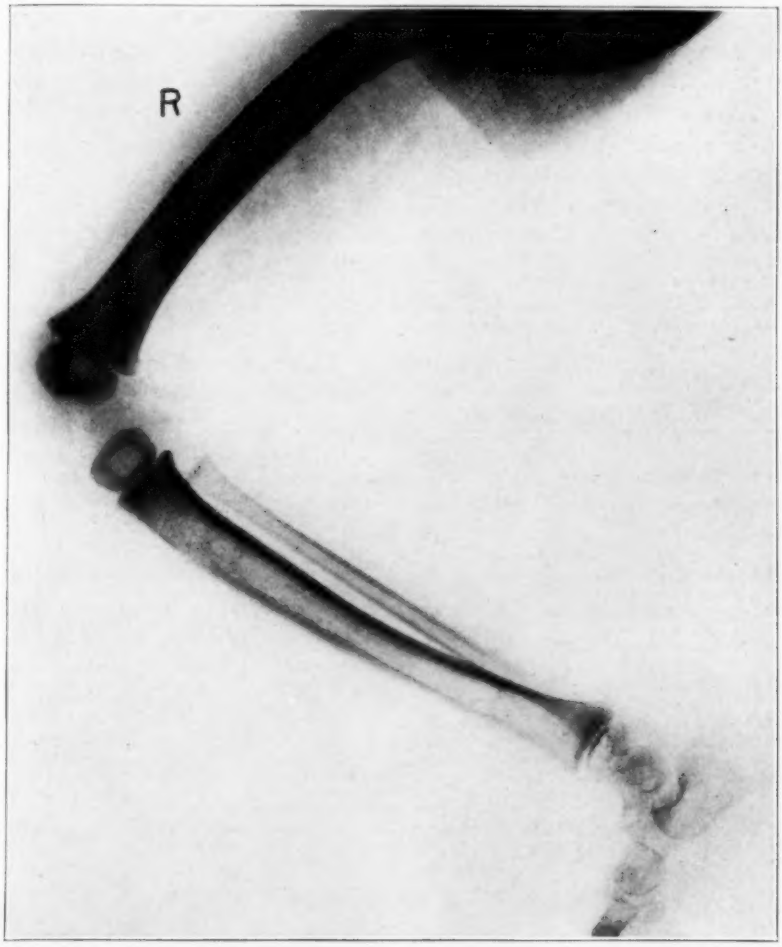

Fig. 53 (C. R.).—Lateral view of left femur, taken on same date as figure 51, showing exaggeration of anterior bowing of the femur and transverse calcified lines in the tibia.

where the hematoma originally was largest may persist for more than a year, sometimes causing a bowing of the shaft; and when there has been epiphyseal separation, the mechanical requirements of the bone, which demand that a new medullary canal shall be carved out from the old bone in line with the new position of the displaced epiphysis, are

such that even after the lapse of two years one may still detect abnormal curvature of the bone or thickening of the cortex on one side (figs. 52, 53 and 54).

Frank described a bony residuum of scurvy in the form of a lateral extension of the edge of the metaphysis in the shape of an anvil or the brim of a hat (fig. 33), which she found persisting for as long as seventeen months after the acute stage of the illness. We have confirmed her observation in three instances in our series, but in roentgenograms of patients who have never been suspected of having scurvy we have occasionally found examples of this picture; so that we are led to doubt its specificity.

TABLE 1.—*Ages on Admission to Hospital*

Age on admission, months	6	7	8	9	10	11	12	13	14	15	16	17	18
Number of cases	1	2	10	12	13	4	5	0	2	1	0	0	1

TABLE 2.—*Data of Duration of Symptoms and Signs*

	Before Admission			After Admission		
Symptom	No. of Cases	Percentage of Total	Range of Duration	No. of Cases	Percentage of Total	Physical Sign
Tenderness	40	78	1 day to 3 months	43	84	Tenderness of extremities
Fever	6	12	1 day to 1 week	38	75	Fever
Swelling of extremities	15	29	1 day to 4 months	36	71	Swelling of extremities
Changes in the gums	5	10	5 days to 3 months	33	65	Changes in the gums
Characteristic attitude	2	4	1 week	29	57	Scorbutic attitude
Skin eruption	2	4	2 to 5 weeks	18	35	Maculopapular eruption
Bloody urine	1	2	3 weeks	18	35	Hematuria
Hemorrhages in other regions*	8	16	1 week to 3 months	10	20	Hemorrhages in other regions*
Weakness of extremities	7	14	1 week to 4 months	7	14	Marked pseudoparalysis

* I. e., other than in the extremities or gums, or in these cases chiefly about the orbit, into the gastro-intestinal tract, or under the periosteum of the skull.

OBSERVATIONS IN AUTHORS' CASES

The ages of our patients on admission to the hospital are presented in table 1.[18] Thirty-five, or 69 per cent, were 8, 9 or 10 months of age, the period of election. The symptoms described in the history, with

18. The tabular surveys represented by tables 1, 2 and 3 cover an analysis of fifty-one patients who entered the hospital at the time they had active scurvy. Another patient, who in his acute illness in infancy was under the care of Dr. Frank McLean and who was treated at home, has not been included in these but has been included in table 4. At the age of 13 years he still showed roentgenographic evidence of the disease in the epiphyses. Dr. George B. Bader assisted us in obtaining a follow-up examination of this patient.

their duration, and the signs elicited on physical examination at the time of admission are given in table 2. In six cases from a small group in which daily examinations of the urine were made, red blood cells persisted in the urine for six days after antiscorbutic treatment had been instituted, and in three cases hematuria lasted fourteen days. All of

Fig. 54 (C. R.).—Lateral view of left femur, taken on same date as figure 51, showing exaggeration of anterior bowing of the femur, with irregularities of the cortical shadow and persistent thickening.

these patients were given 3 ounces (90 cc.) of orange juice daily as a routine. Persistence of hematuria of this kind has been mentioned by Hess.[19] In eighteen cases in which determinations of blood calcium

19. Hess, A.: Scurvy, Past and Present, Philadelphia, J. B. Lippincott Company, 1920.

and phosphorus were made early in the period of hospitalization, the average calcium content of the serum was 9.2 mg. in 100 cc., with a range of from 7.3 to 11.7 mg. The average inorganic phosphorus was 3.3 mg. in 100 cc. of serum, with a range of from 1 to 6.2 mg. Table 3 indicates the number of patients who on successive days following admission showed fever for which no cause other than scurvy was demonstrable. The threshold of fever for this tabulation was taken as 100 F. (37.8 C.), but an attempt was not made to tabulate the degree of fever in individual cases.

Table 4 gives, week by week, the incidence of positive roentgenologic manifestations in our series. The figures for percentage reported refer to the proportion of positive signs with respect to the number of x-ray examinations made during the period of time covered in each column. In addition to the signs of active and healing scurvy which were enumerated at the beginning of this article, we have included in the tabulation certain of the manifestations of active rickets, namely, "cupping" or broadening of the epiphyses and "fraying" of the ends of

TABLE 3.—*Number of Patients with Fever*

Day of treatment	1	2	3	4	5	6	7	8	9	10
Number of patients with fever	37	33	32	21	16	10	6	4	4	4

the long bones. It was found necessary to take the rachitic factor into account, since its presence modifies the scorbutic picture to a considerable degree, as already described. It is likewise true, however, as Wimberger has pointed out, that the presence of severe scurvy greatly modifies the usual roentgenologic picture of rickets. From a clinical point of view, the majority of our patients—all, in fact, except those markedly below weight for their age—showed the physical signs of rickets; and we feel that we have in all probability erred on the side of conservatism by using only the x-ray reading for the diagnosis of rickets in the compilation of this table.

It is evident from table 4 that in the first week of observation and antiscorbutic treatment the roentgenologic manifestations that occurred most frequently, according to our reading of the films, were the increase in width and density of the zone of preparatory calcification and the presence of the adjacent zone of rarefaction. Other signs which were of moderate constancy at this stage and which occurred in from 35 to 65 per cent of the examinations, were the broadening and intensity of the periphery of the centers of ossification and the area of rarefaction at their centers, separation of the epiphyses, spurs and calcification of elevated periosteum. The large number of transverse calcified lines reported in the first week column, as well as in those of succeeding weeks, means that lines were seen and reported as such; but an attempt

TABLE 4.—*Data of Roentgenologic Examination*

Time elapsed since commencement of treatment*	Weeks												Years					
	1	2	3	4	5	6	7	10	15	20	35	50	1½	2	3	4	10	12
Broadened zone of preparatory calcification	35	16	9	6	2	0	2	0	0	0	0	0	0	0	0	0	0	0
	83%	76%	69%	67%	67%	0%	100%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Spurs	26	12	5	3	1	0	0	0	0	0	0	0	0	0	0	0	0	0
	62%	57%	38%	33%	33%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Zone of rarefaction	35	16	11	5	2	0	0	0	0	0	0	0	0	0	0	0	0	0
	83%	76%	84%	56%	67%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Center of Ossification: Intensity of periphery	24	16	10	8	3	3	2	1	0	1	0	0	0	0	0	0	0	0
	57%	76%	77%	89%	100%	100%	100%	50%	0%	33%	0%	0%	0%	0%	0%	0%	0%	0%
Center of Ossification: Wide peripheral zone	23	14	8	7	2	3	2	1	0	1	0	0	0	0	0	0	0	0
	55%	67%	62%	78%	67%	100%	100%	50%	0%	33%	0%	0%	0%	0%	0%	0%	0%	0%
Center of Ossification: Rarefaction at center	25	15	11	8	2	3	2	1	0	1	0	0	0	0	0	0	0	0
	60%	71%	84%	89%	67%	100%	100%	50%	0%	33%	0%	0%	0%	0%	0%	0%	0%	0%
Separation of epiphysis	19	12	10	5	3	2	0	1†	0	2†	1†	0	0	0	0	0	0	0
	45%	57%	77%	56%	100%	100%	0%	50%	0%	67%	50%	0%	0%	0%	0%	0%	0%	0%
Clouding of trabeculae	8	3	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	19%	14%	23%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Thinning of cortex	12	7	5	3	1	1	0	0	0	1	0	0	0	0	0	0	0	0
	29%	33%	38%	33%	33%	33%	0%	0%	0%	33%	0%	0%	0%	0%	0%	0%	0%	0%
Hemorrhage into soft parts	11	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	26%	14%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Subperiosteal fracture	1	2	1	2	1	0	0	0	0	0	0	0	0	0	0	0	0	0
	2%	10%	8%	22%	33%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Calcified elevated periosteum	15	11	12	9	3	2	0	2	1	1	1	0	0	0	0	0	0	0
	36%	52%	92%	100%	100%	67%	0%	100%	100%	33%	50%	0%	0%	0%	0%	0%	0%	0%
Transverse calcified lines	10	3	3	1	1	2	1	2	0	3	2	1	1	2	1	0	0	0
	24%	14%	23%	11%	33%	67%	50%	100%	0%	100%	100%	100%	25%	67%	100%	0%	0%	0%
Central scar in center of ossification	0	0	0	0	0	1	0	0	0	2	2	1	4	3	1	1	1	2
	0%	0%	0%	0%	0%	33%	0%	0%	0%	67%	100%	100%	100%	100%	100%	100%	50%	100%
Thickened shaft	0	0	0	0	0	0	0	0	0	0	0	0	1	3	1	0	0	0
	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	25%	100%	100%	0%	0%	0%
Curvature of shaft	0	0	0	0	0	0	0	0	0	0	0	0	3	2	1	0	0	0
	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	75%	67%	100%	0%	0%	0%
Anvil deformity (Frank)	0	0	0	0	0	0	0	0	0	0	1	1	2	0	0	0	0	0
	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	50%	100%	50%	0%	0%	0%	0%	0%
Cupped or broadened epiphyses	30	17	9	6	3	3	1	0	0	1	0	0	0	0	0	0	0	0
	71%	81%	69%	67%	100%	100%	50%	0%	0%	33%	0%	0%	0%	0%	0%	0%	0%	0%
Frayed epiphyses	25	11	9	4	2	0	0	0	0	0	0	0	0	0	0	0	0	0
	60%	52%	69%	44%	67%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Examinations made	42	21	13	9	3	3	2	2	1	3	2	1	4	3	1	1	2	2

* Column 1 indicates observations during the first week; column 10 indicates observations from the end of the seventh week to the end of the tenth week, etc.; column 1½, years covering the period from the end of the fiftieth week to the end of the eighteenth month.

† Fused to shaft in new position.

was not made to single out and report only the lines caused by scurvy. The frequency with which one encounters such lines due to a variety of causes in x-rays in the average pediatric hospital service is familiar. Lines were not detected in photographs made after the lapse of three years from the commencement of treatment. The column for the first week also shows that other signs of acute scurvy which are at times helpful in differential diagnosis, such as clouding of the trabecular structure

Fig. 55.—I. F., age on admission, 10 months. Swelling and tenderness of both lower extremities, four or five weeks. Clinically he showed severe scurvy, with separation of both lower femoral epiphyses. He remained in the hospital eight weeks. Since discharge he has had no disease or injury that would be considered to produce x-ray changes in the bones. Condition eleven years and ten months after commencement of treatment for scurvy. A central scar of rarefied bone, surrounded by a faint ring of dense bone, persists in the upper epiphysis of the tibia.

of the shafts, thinning of the cortex and hemorrhage into the soft parts, were found by us in less than 30 per cent of the examinations.

Study of the next six columns in table 4 shows that the characteristic changes in the zone of preparatory calcification may persist for

from five to seven weeks; in the zone of rarefaction for five weeks; spurs from two to five weeks; clouding of trabeculae for three weeks; hemorrhage into the soft parts for one or two weeks, and thinning of the cortex for as long as twenty weeks. Some of the signs of the acute stage of the disease merge into those of the stage of healing listed lower down in the table. Thus, the broadened zone of preparatory calcification becomes a transverse calcified line; spurs disappear because of calcification of the elevated periosteum; the zone of rarefaction is obliterated by the reappearance of prominent trabeculae, and with growth of the centers of ossification the characteristic acute changes found here give place to the condition listed as the central scar in centers

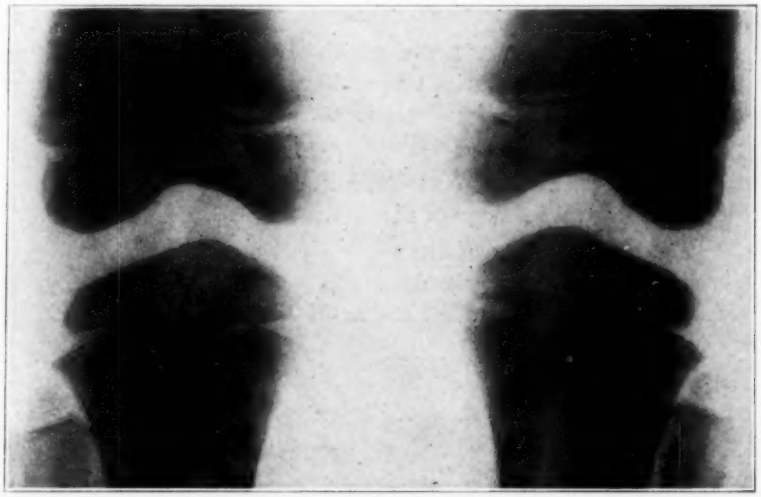

Fig. 56.—B. S., aged 6 years, anemia "von Jaksch's" type. No history suggestive of scurvy. Splenectomy at age of 2; x-ray at 4 years of age. There are central scars of rarefied bone somewhat resembling scorbutic scars in the upper epiphyses of both tibia and lower epiphyses of both femora.

of ossification. Calcification of elevated periosteum may in some cases be distinguishable as long as thirty-five weeks after commencement of treatment; and if the hematoma has been extensive, it will have left its mark after from one and a half to three years as unilateral thickening of the shaft, at times with accompanying curvature. The last sign to be found is the kernel of rarefied bone, surrounded by a thin shell of dense bone, in the centers of ossification.

Disturbances of general nutrition may alter the rate, though not the general order, of these changes. Thus, one patient who contracted a severe form of bacillary dysentery at the time he had acute scurvy and who for several weeks of his stay in the hospital remained at practically

a stationary weight and in a state of imminent if not actual dehydration grew so slowly during that period that, even twenty weeks after the commencement of treatment for scurvy, the centers of ossification still presented the picture usually seen during the first week or two of average healing, and the cortex of the long bones was still thin. On the other hand, a second patient with severe rickets in addition to severe scurvy, whose epiphyses, at the time she was first seen, may already have formed a broad zone of uncalcified bone matrix, showed such rapid apparent growth during the first six weeks of the period of observation in the hospital that at the end of that time the rarefied centers of the centers of ossification were enclosed in a thick shell of newly formed bone.

SUMMARY

1. The types of disturbances which may be found in roentgenograms of the long bones in infants with scurvy have been enumerated, and their modulations during the process of healing described.

2. A series of 115 roentgenologic examinations in 52 patients with scurvy are analyzed, with particular reference to the variation in the picture according to the stage of the disease.

3. The relative value of the x-ray signs of scurvy from the point of view of diagnosis in acute cases and the relative frequency of the various signs presented for all stages of the healing process for our series are discussed.

4. The period covered by this analysis ranges from the first day of active antiscorbutic treatment to twelve years later.

5. The series is briefly analyzed from the point of view of symptomatology and physical manifestations.

THE PREVENTORIUM CHILD

A CLINICAL REVIEW OF THREE HUNDRED CASES*

I. D. BRONFIN, M.D.

DENVER

The literature on the so-called pretuberculous child, the child with juvenile tuberculosis, tracheobronchial adenopathy and occult or latent tuberculosis, has grown in the last decade to such tremendous proportions that it is almost beyond the task of one or even of a group of workers to cover the subject in its entirety. It is no longer necessary to stress the important fact that tuberculous infection starts during early infancy and increases in incidence as age advances, and that from 60 to 80 per cent of children who attain the age of puberty harbor the cause of the disease. That only proportionately few succumb to the ravages of clinical tuberculosis in adult life should not minimize the problem of tuberculosis, since relatively little is known of the factors which determine the future course of tuberculous infection. Whether a tuberculous lymph node located near a blood vessel or bronchus will caseate and empty its contents into the lumina of its neighbors, thereby producing miliary tuberculosis or bronchopneumonia, or whether it will become fibrosed and calcified and imprison, perhaps forever, the offending organism, is a matter of speculation. From available data, it is known that the exanthems and acute respiratory infections not infrequently cause a reactivation of a dormant focus which progresses to clinical tuberculosis. It is likewise important not to ignore the rôle which the economic factor may play in determining the outcome of this ubiquitous infection. The rapidly increasing number of new outbreaks of adult tuberculosis in some of the European countries during the World War further emphasizes the necessity of directing attention to the intensive study of infected children. It is generally conceded that if tuberculosis as it is found among children, even in its most benign forms, were detected in time and given the vigilant supervision it deserves in the present generation, the incidence of adult clinical tuberculosis in the next generation would be greatly reduced.

The familiar "contact" case can best crystallize the problem. The clinician knows that such a child, having lived in a more or less intimate association with one suffering from active pulmonary tuberculosis, has

* Received for publication, June 19, 1928.

* From the National Jewish Hospital and the University of Colorado School of Medicine.

* Read at the Joint Meeting of the El Paso County Sanatorium Association and the Denver Sanatorium Association, April 21, 1928.

become infected. The positive tuberculin reaction constitutes the indisputable evidence, but, as is often the case, the child is well nourished and apparently free from active disease. In the majority of instances, such a child grows to maturity and is unhampered throughout life by the latent infection. The latter may possibly have even been the background for immunity against adult tuberculosis, but the converse, which is the great bugbear of all intelligent physicians and parents, is apt to take place, namely, the insidious or sudden onset of some obscure ailment which eventually proves to be miliary tuberculosis or tuberculous meningitis. It is a matter of common knowledge that the child who is

TABLE 1.—*Principal Complaints on Admission*

	No.	Per Cent
Underweight	93	31
Underweight with fatigue	35	11.67
Underweight, fatigue and irritability	18	6
Irritability alone	10	3.33
Frequent colds and fever	40	13.33
Cough	20	6.67
Capricious appetite	30	10
Periodic swelling of glands of neck	21	7
Spinal deformities, deformities of the joints or local pain	18	6
Unclassified (mostly a combination of many symptoms)	15	5

TABLE 2.—*Principal Physical Manifestations*

	No.	Per Cent
Underweight (7 per cent or more)	210	70
Cervical and submaxillary glandular enlargements	135	45
Osseous tuberculosis	18	6
Scoliosis	3	1
Definite pulmonary tuberculosis (latent, quiescent or active)	6	2
Suspicious signs of tracheobronchial adenopathy	30	10
Bronchial asthma	10	3.33
Chronic bronchitis?	3	1
Valvular heart disease	12	4
No apparent abnormality other than subjective symptoms with or without underweight	83	27.66

known to be infected often manifests symptoms of impending danger, but these symptoms are of such transient nature and are so uncharacteristic that they are usually attributed to other causes, such as the common cold, grip and intestinal indigestion. The more alert physician may consider these manifestations as tuberculous in origin, but owing to the long intervals during which the child is well, he loses track of the case until the child appears ten or fifteen years later in a terrified mental state to report the dramatic story of an initial hemoptysis or a train of symptoms which prove to be due to advanced and active pulmonary tuberculosis. What has taken place in the interim? Has that child manifested, during that period of apparently perfect health, any deviations from the normal which, if observed, could have been interpreted as warnings to avert a tragedy? It is this unexplored gap between infection and disease that deserves the greatest study, and it

was with this object in view that a study of case records of 300 children in the preventorium was undertaken.

The Hofheimer Preventorium of the National Jewish Hospital opened its doors in October, 1920. Up to Feb. 20, 1928, at the time this survey was begun, it had cared for 300 children recruited from every state in the Union. The ages ranged from 2½ years to 16 years, the average being 9.35 years. The average duration of residence was 290 days. There was a slight preponderance of males, 56.6 per cent, as against 43.4 per cent females. It is regrettable that during the early period of the preventorium's existence some essential clinical routine, such as complete history taking and tuberculin tests, was not regularly done. In general, however, sufficient data were obtained, an analysis of which discloses some interesting facts.

TABLE 3.—*Comparative Clinical Symptoms in Tonsillectomized and Nontonsillectomized Children*

	Tonsillectomized 159, or 53 Per Cent		Nontonsillectomized 141, or 47 Per Cent	
	Number	Per Cent	Number	Per Cent
Underweight	110	69.1	100	70.9
Exposure	81	50.9	74	52.4
Catarrhal	94	59.1	86	60.9
Cough	15	9.4	6	5.3
Cervical adenopathy	64	40.2	71	50.3
Tracheobronchial adenopathy	20	12.5	10	7.1
Osseous tuberculosis	10	6.3	8	5.6
Pulmonary tuberculosis	4	2.5	2	1.4
Bronchial asthma	4	2.5	6	4.2
Chronic bronchitis	1	0.6	2	1.4
Valvular heart disease	4	2.5	6	4.2

EXPOSURE, TONSILLECTOMY AND CERVICAL ADENOPATHY

To evaluate the clinical significance of palpable or visible cervical glandular enlargement, the children were classified into tonsillectomized and nontonsillectomized groups after infections of the teeth and sinuses were excluded. Only in those who had lived in a home in which a member of the family suffered from active pulmonary tuberculosis was the condition classified as due to exposure. Those giving a history of having had more than three attacks of acute infections of the upper respiratory tract during the year were classified as catarrhal cases. Cough as an important symptom was considered only when it persisted for six weeks or longer after the disappearance of the acute catarrhal manifestations.

It can readily be seen from table 3 that tonsillectomy did not appreciably modify symptoms or clinical manifestations interpreted as possibly being due to tuberculosis, although the purely nontuberculous conditions, notably the chronic respiratory and cardiac conditions, were more prevalent in the nontonsillectomized group.

CLINICAL EVALUATION OF CERVICAL ADENOPATHY

Under this term is included all glandular enlargements in the region of the neck, namely, the superficial cervical, the upper and lower chain of the deep cervicals, the submaxillary and the submental. An arbitrary classification based on size was resorted to in order to simplify the records. Glands the size of a pea were recorded as plus 1; those the size of a small bean, plus 2, and those the size of a lima bean or larger, plus 3. Glands smaller than a pea, even if readily palpable on both sides, were recorded simply as "glands palpable" but were not considered pathologic. The 135 children with glandular enlargement were examined during their residence in the hospital in the otolaryngologic and dental departments, in which those with recognizable foci of infection received the indicated treatment. In not a few there was a gradual recession in the size of the glands, but in most cases a notable decrease was not noted after regional foci of infection were removed.

The difficulty of making a differential diagnosis between chronic tuberculous and chronic nontuberculous cervical adenitis is well known. However, the fact that the incidence of adenopathy was not appreciably greater in the nontonsillectomized than in the tonsillectomized group points to the probability that in at least a considerable number the glandular enlargements were possibly due to tuberculous infection. Furthermore, as will be seen from table 4, only 19 per cent with pathologic glandular enlargement were noted in the nonexposed group, whereas in the exposed group the percentage was more than 62.

EXPOSURE TO INFECTION

On the basis of exposure to infection, the cases were classified into three groups. Of the total number, 155, or 51.6 per cent, had been subject to an intimate exposure for a period of six months to nine years. When the history was not definite but suggestive, such as the presence of a long standing respiratory infection in a member of the family or the temporary residence in the family of one who was suspected of tuberculosis or in whom the condition subsequently was diagnosed as such, the classification was that of probable exposure. In this group there were 63, or 21 per cent. A negative history was obtained in 93, or 31 per cent.

A comparative study of the symptomatology as observed during the residence in the hospital and the principal physical symptoms of these three groups was deemed essential. Diurnal variations in temperature of 1.5 F. were not considered abnormal in the absence of other symptoms. When this slight febrile rise was accompanied by a subjective sensation of heat, headache or malaise, it was recorded as fever. Greater significance was attached to periodic rises in tempera-

ture of 100 F. or over, even when other symptoms were not present. Any child who, during a given month, had two or more recurrences of fever in the afternoon, lasting from one to two days, was classified as a fever case after focal infection was excluded with reasonable certainty. As the hospital maintains its own grammar school, in charge of a licensed teacher with extensive experience in nursing persons with tuberculosis, there was ample opportunity to observe the children closely with reference to fatigue and irritability. Cough as a definite symptom was recorded, as previously stated, only when it persisted for a long time and especially when it occurred during the night and disturbed the child's sleep. The habit cough was easily recognized and in most instances could be overcome by proper training. Incidentally, it is noteworthy that only this symptom was accurately observed and evaluated by the parents or guardians, as in twenty cases cough was

TABLE 4.—*Comparative Symptomatology and Clinical Manifestations in "Contact" and "Probable Contact" Cases and Noncxposed Cases*

	Definite Exposure 155, or 51.6%		Probable Exposure 93, or 31%		Negative Exposure 52, of 17%	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Fever	75	48.38	9	9.67	6	11.5
Fatigue	85	54.83	15	16.12	10	19.2
Irritability	58	37.41	16	17.2	6	11.5
Cough	16	10.3	4	4.3	1	1.9
Underweight	150	96.8	44	47.3	16	30.8
Cervical adenopathy	97	62.6	28	30.1	10	19.1
Tracheobronchial adenitis	18	11.6	9	9.6	3	5.8
Osseous tuberculosis	14	9	2	2.1	2	3.9
Pulmonary tuberculosis	3	1.9	2	2.1	1	1.9

given as the principal reason for suspecting illness, and in only twenty-one cases in the entire series was persistent cough observed.

An analysis of table 4 yields convincing proof of what is generally accepted though not sufficiently stressed, namely, that intimate exposure not only invariably causes infection, but ultimately produces active disease in many children. The symptoms are, as a rule, vague and ephemeral and hence are readily overlooked by the average parent. While the classic picture of listlessness, fatigue, irritability and capricious appetite was often associated with fever, there were many children who had to be restrained from activity at a time when they had a temperature of 100 F. It may be argued that fever in the absence of other symptoms is not an indication for active treatment. This is true of children in whom tuberculosis can be excluded with some degree of accuracy, but in the group of children detailed here—and this is the type of child present in many homes and in every community—the frequently recurring fever without any other subjective complaint and lasting from twenty-four to forty-eight hours should be considered of great

significance, especially in the children who have been definitely exposed, even when the physical and roentgen-ray examinations give negative results.

Fever unaccompanied by any other subjective symptoms is not an uncommon manifestation in adult pulmonary tuberculosis. It is a common experience of many clinicians to detect a temperature of 101 F. or higher in ambulant patients. Such patients often refuse to discontinue work, for they claim to be in their usual state of health, except for some increase in respiratory symptoms, such as cough and expectoration or slight pleuritic pain. The penalty that such patients pay, if they continue pursuing their occupations while they have fever, is well known, but comparatively few clinical observations have been made on the child with fever, whose sensorium is not developed and who therefore is much more unaware of such symptoms as fever or fatigue.

It should be noted that of the ninety children with fever, in seventy-five, or 83 per cent, it occurred in the definitely exposed group as against

TABLE 5.—*The Tuberculin Reaction in Ninety Children*

		Reaction			
		Positive		Negative	
	No. of Cases	Number	Per Cent	Number	Per Cent
Definite exposure	20	19	95	1	5
Pobable exposure	40	28	70	12	30
Negative exposure	30	16	53	14	46.6

nine, or 1 per cent, in the doubtful group and only six, or 0.6 per cent, in the negative group. In other words, fever and fatigue were noted in about 50 per cent of the known "contact" cases, and glandular enlargements of the neck in more than 60 per cent.

THE TUBERCULIN REACTION

It is realized that much is detracted from the value of this report because of a failure to perform the tuberculin test as a routine in all cases. The test was made on only ninety children. In eighteen the Pirquet method was employed, and seventy-two had the intradermal test. Table 5 refers to the three groups depicted in table 4. This table shows in a striking manner the effect of exposure and its bearing on infection.

Of the twenty-seven children in whom the reaction was negative, in sixteen the Pirquet test only was made. It is quite likely that if this test had been followed by the intradermal test a greater percentage of positive reactions would have been recorded. It is also plausible to assume that those figures are a fair index to what probably would have been the result if all children had been tested.

PHYSICAL SIGNS

In common with many other observers, my associates and I were impressed with the paucity of physical symptoms in many children whose history, clinical course, roentgen-ray picture and tuberculin reaction pointed to tuberculosis. Conversely, there were many who gave classic signs of a tracheobronchial tuberculosis on both physical and roentgen-ray examinations, but who failed to show any evidence of tuberculosis clinically. The D'Espine sign proved misleading, as was also interscapular dulness. In about 50 per cent of those who gave these physical signs, the roentgen-ray films failed to show glandular enlargements within the chest. In only 5 per cent of the entire group were dilated veins on the chest or abdomen noted. This observation, however, is significant, as twelve of the fifteen children with dilated veins showed unmistakable evidence of tracheobronchial tuberculosis. Faulty posture, stressed by some writers, was noted in only 10 per cent of the series. The long, slender, so-called asthenic type of chest was observed in more than 25 per cent of the patients, but positive symptoms in this group were not greater than in those with normal chests and well developed musculature.

ROENTGEN-RAY OBSERVATIONS

This part of the study was approached with considerable caution, since we were fully aware of the pitfalls in diagnoses likely to occur from misinterpretation of the films. The current literature on this subject was reviewed and, as a further precaution, two roentgenograms, characteristic of a great many that have been encountered here, were sent to Dr. F. Maurice McPhedran of the Phipps Institute for his opinion on discrete circular densities, irregular and somewhat less dense shadows, enlarged hila and primary foci. The information received proved helpful.[1]

The plates were studied by serial number,[2] without any knowledge of his history or clinical symptoms in any given case. Preconceived notions were thus avoided. The nomenclature used in the classification, based in part on the recommendations made by the Committee of the National Tuberculosis Association, is as follows: (1) normal; (2) enlarged hila; (3) calcification at hila; (4) primary foci; (5) tracheobronchial tuberculosis and (6) pulmonary tuberculosis.

Of the total group, 72, or 24 per cent, were normal; 228, or 86 per cent, showed enlarged hila; 195, or 65 per cent, manifested calcification

1. Acknowledgement is hereby made to Dr. McPhedran for his assistance.

2. Acknowledgement is hereby made to Dr. Louisa T. Black, clinical pathologist and roentgenologist of the hospital, for the valuable technical assistance and suggestions in this part of the work.

in the hilar regions; 66, or 22 per cent, showed tracheobronchial glandular enlargement; in 35, or 11.6 per cent, were noted primary foci, and in 34, or 11.3 per cent, frank pulmonary tuberculosis was found.

In reviewing the plates, we had in mind certain basic principles elaborated by clinicians and roentgenologists. A normal hilum shadow, cast mainly by the pulmonary vessels and bronchi, appears as an oblong, faintly dense opacity, which rapidly loses the opaqueness as it extends laterally and finally fades into the lung field. It has indefinitely

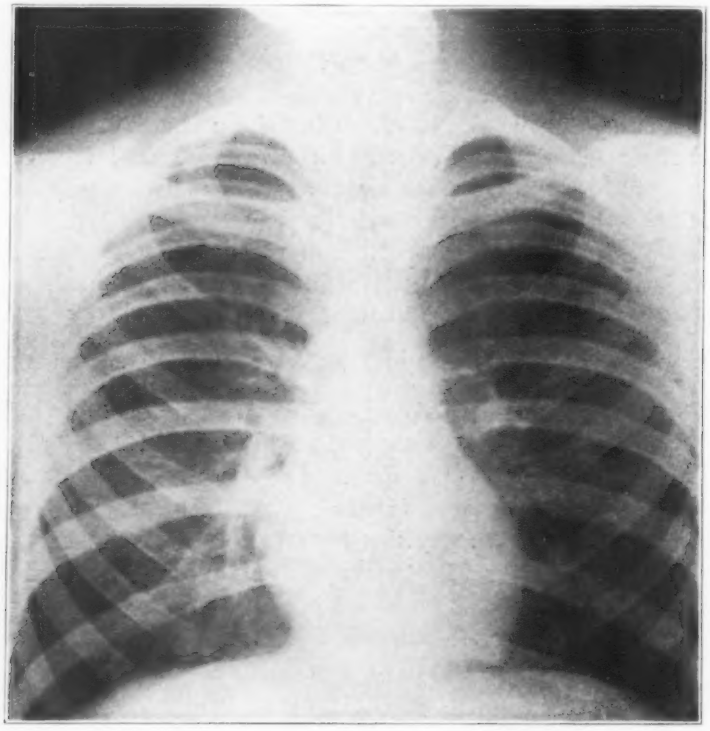

Fig. 1.—Roentgenographic appearance of normal chest, a boy, aged 10 years, referred with a diagnosis of tuberculous enterocolitis. The colitis proved to be due to Endameba histolytica. The tuberculin reaction was negative. There was no history of exposure. The appearance of the normal hila and the air-bearing tissue medial to the opacities cast by the blood vessels should be noted.

circumscribed margins and stems and small pear-shaped or circular shadows of somewhat greater density. These pear or dumbell-shaped densities are cast by blood vessels radiated axially or obliquely, as has been demonstrated by McPhedran.[3] At times, a circular shadow con-

3. McPhedran, Maurice F.: The Diagnosis of Tracheobronchial Tuberculosis, Am. J. M. Sc. **173**:245 (Feb.) 1927.

taining air can be seen near the density cast by the blood vessel. It is readily recognized as an axially radiated bronchus.

In the normal hilum, especially on the right, a narrow, vertical, air-bearing strip is seen running parallel to and medial to the hilum shadow. Baum [4] said that the degree of abnormality of the hilum can be estimated by the extent of obliteration of this air-bearing space. It is important, however, to guard the patient against the slightest rotation while the film is taken, for such malposition will often obscure this interspace, and the hilum will be interpreted as pathologic.

When the film in the series reported showed an increased vascularity, especially over the inner and middle pulmonic zones, it was set aside for

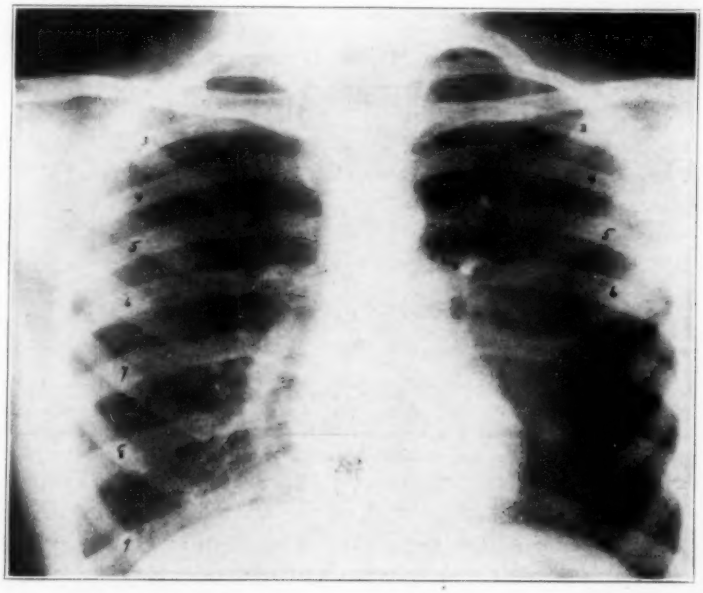

Fig. 2.—Roentgenographic appearance of chest of girl, aged 8 years, with a history of probable exposure. The tuberculin reaction was positive. There were no symptoms except slight underweight. The normal hila, the axially radiated bronchus *A*, the axialy radiated blood vessel *B*, and immediately below the vessel a shadow of greater density *C*, which is a calcium-bearing lymph node—the only roentgenographic evidence of tuberculous infection—should be noted. The circular density *D*, commonly mistaken for a lymph node, is also an axially radiated vessel.

a review of the clinical record to determine whether an acute respiratory infection was present at the time the film of the chest was obtained. Such roentgenograms and also those of patients found to be suffering from bronchial asthma or intercurrent acute infections were considered

4. Baum, Felix: A Roentgen-Ray Phenomenon in the Normal and Pathological Hilum, J. A. M. A. **88**:634 (Feb. 26) 1927.

as normal for that clinical entity, unless subsequent films showed a decrease in the vascular markings, and the hila continued to be abnormally enlarged. An important flaw in the roentgenologic study of these cases is the fact that in only eighty-four cases were serial films made. Oblique or lateral exposures were made in only six cases. There is no doubt that if oblique films had been made as a routine, a larger percentage of tracheobronchial involvement would have been demonstrated.

Table 6 illustrates in a striking manner the importance of roentgenography as an aid in the diagnosis of latent or active tuberculosis in children. If one disregards the questionable significance of enlarged hila, calcification has to be considered as a most probable evidence of tuberculosis. To be sure, calcification of lymph nodes in the adult has little clinical importance, but if one takes into consideration the fact that the average age of the children in this series was less than 10 years,

TABLE 6.—*Roentgen-Ray Examinations in Three Hundred Preventorium Children*

Cases	Normal		Enlarged Hilum		Calcification at Hilum		Tracheobronchial Tuberculosis		Primary Foci		Pulmonary Tuberculosis	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
155 Definite exposure.....	6	8.3	178	78	148	75.9	54	81.8	26	74.3	27	79.3
93 Probable exposure.....	12	16.7	35	15.4	37	19	9	13.6	9	25.7	6	17.8
52 Negative exposure.....	54	75	15	6.6	10	5.1	3	4.6	0	....	1	2.9
Total	72	100	228	100	195	100	66	100	35	100	34	100

the presence of calcification cannot be entirely ignored. Furthermore, since lymph nodes cannot be seen on the roentgen-ray film unless they project to one side of the bronchovascular stem and contain a deposit of calcium, it is obvious that only parts of the nodes are seen on the film. The exact size of the lymph nodes or their exact pathologic condition is rarely determined. The faintly granular appearance of some of these nodes suggests caseation in addition to calcification. As will be seen from the case reports that follow and the accompanying figures, the symptoms could not be attributed to any other clinical entity except tracheobronchial tuberculosis.

The discrepancy between physical and roentgen-ray manifestations is also marked in those with pulmonary involvement. Fifteen of the eighteen children with involvement of the bones showed pulmonary lesions, and only two gave definite physical signs. The incidence of pulmonary involvement in extra-pulmonary tuberculosis in our series is not surprising and is in accord with the observations made by Lo Grasso.[5]

5. Lo Grasso, H.: Heliotherapy in Tuberculosis, N. Y. State J. Med. **22**:281 (June) 1927.

CLINICAL COURSE

During Residence.—The temperature subsided to within normal variations in sixty-eight of the ninety patients with fever, or 74.4 per cent, after three months of routine treatment. Recurrence of fever was noted in nine, or 13 per cent, in from six to ten weeks after restrictions on physical activity were removed. Fatigue and irritability decreased in proportion to the general improvement, although it is realized that the degree of those symptoms, especially fatigue, was never accurately determined. The observations were made by the nurses and teacher, who would record in a given case, "does not tire as easily" or "child is more playful and not as quarrelsome." Weight was a much more reliable index. Only eighteen children failed to gain in weight. Of these, eight were "contacts," five probable "contacts," and five were of the nonexposed group. Seven of these eighteen children had osseous tuberculosis of moderate severity; five showed roentgenographic evidence of tracheobronchial adenopathy, and in the remaining six significant pathologic changes were not demonstrated either by physical or roentgen-ray examinations.

Normal weight was attained in only 70 per cent of the total. Of the ninety cases in which the patients remained underweight at the time they left the preventorium, sixty-four, or 71.1 per cent were "contact" cases, twelve, or 13.3 per cent, were probable "contact" cases, and fourteen, or 15.5 per cent, were of the nonexposed group. In eighty-four, or 93.3 per cent, of those who were underweight at the time of their discharge, there were present symptoms and physical and roentgen-ray signs warranting a diagnosis of glandular, osseous or pulmonary tuberculosis.

A definite decrease in the glandular enlargements of the neck was noted in only 24 cases of the total series of 135, or 17.7 per cent, despite intensive heliotherapy. Noteworthy changes were not noted in any of the 84 serial roentgen-ray plates, except in those of patients who had parenchymal infiltrations. In the 34 patients whose roentgenograms showed pulmonary lesions, follow-up plates were made of 8. Of those, 5 did not show changes, 2 showed a clearance and one showed an increased involvement.

After Discharge.—The difficulties of following up patients clinically after they leave the institution are familiar to every sanatorium worker. So far, fairly reliable data have been obtained in only forty-four cases. The effort is being continued and if it is successful, the data will constitute the subject for a future report. From the information available, it would seem that about 3 per cent of those with primary pulmonary foci and tracheobronchial adenopathy succumb to clinical disease in early adult life.

TREATMENT

The initial observation in each new case covered a period of two weeks. If the child was found to be free from symptoms, permission was granted for him to attend school. Attendance at school is from 9 to 12 in the morning, five days weekly, except during July and August. Those who complained of persistent though slight fatigue and an evening temperature of 99.5 F. were wheeled to school, where they remained for one hour. The period was gradually increased when untoward effects were not noted. Exercise outdoors was not allowed when there was any tendency for the child to tire easily. There was no set rule, however, for not a few patients began to show general improvement and gain in weight when all restrictions were removed, despite some suspicion symptoms. Here individualization plays a most important rôle, much more so than in the treatment of adults.

The gradation method of heliotherapy was employed in all patients, except in those who had a temperature of 101 or more. After the entire body had been insolated to the extent that heliotherapy could be administered for one hour daily, the child was given the "uniform" which consisted of a pair of trunks to be worn all day, except in inclement weather.

The food consisted of a general mixed diet and a quart of milk to be taken with meals. Fresh vegetables and fruit juices were a part of the daily dietary. Contrary to general practice, nourishment was not served between meals, except to those with capricious appetites. One glass of milk was served shortly before the children retired. Experience has taught that under the regulated life which is afforded them, the children thrived best on three standard meals.

Compulsory hours for sleep are from 1 to 3 in the afternoon. From 3 to 5 are the play hours on the recreation grounds under the supervision of the teacher or nurse. A thirty minute rest period is insisted on before each meal. Indoor play in the winter and outdoor play in the summer are permitted after the evening meal for one hour, and all must retire for the night at 8:00 o'clock.

Under this regimen the majority of the children became symptom-free and at the time of discharge seemed in perfect health. Readmission was sought for thirty-eight children from six months to two years after discharge. The principal complaint was underweight, which in most instances was due to poor hygienic environment and mismanagement. This is a grave social and economic problem which must be left for its solution to the social service worker. In only ten cases were there symptoms besides underweight to constitute a medical indication for readmission.

CASE REPORTS

CASE 1.—*Intimate Exposure, Active Tracheobronchial Tuberculosis with Apparent Recovery.*

F. L., a girl, aged 4 years, was admitted to the preventorium on July 6, 1921. At 31, her mother had active pulmonary tuberculosis of three years' duration, with positive sputum. For two and one half years prior to admission, the child had been subject to frequent colds, intermittent fever, irritability and poor appetite. The tonsils and adenoids had been removed one year before admission, without

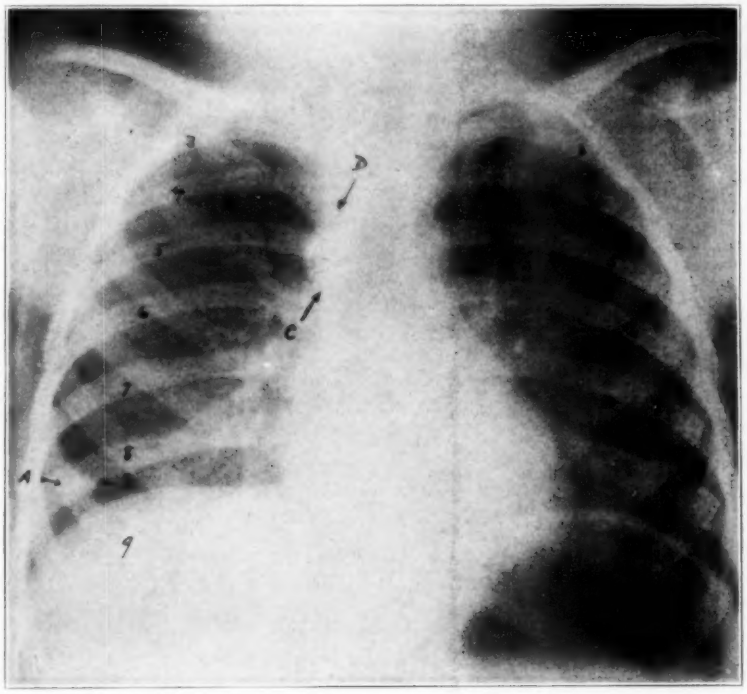

Fig. 3 (case 1).—Primary tuberculous focus *A-B*, and a large calcium-bearing lymph node *C-D*.

any improvement. The principal complaints on admission were: fatigue, occasional fever, frequent colds and underweight. The essential physical signs were: underweight, 11 per cent; tuberculin reaction, positive; anterior cervical glands on the left, plus 2, and slight interscapular dulness on both sides, more marked on the left. A roentgenogram (fig. 3) showed the primary tuberculous focus in the outer zone of the right lung at the upper border of the ninth rib, (*A-B*) and a pear-shaped glandular enlargement (*C-D*), granular in appearance, near the right border of the spine opposite the fifth and sixth ribs. The patient was discharged on Feb. 24, 1923, markedly improved, but remained 4 per cent underweight. The last report stated the child had been living for the last two years in New York City and had been enjoying uninterrupted good health.

CASE 2.—*Intimate Exposure, Intermittent Fever, Intrathoracic Glandular Tuberculosis.*

B. B., a girl, aged 6 years, was admitted to the preventorium July 11, 1927, on account of frequent colds, periodic attacks of fever, irritability and poor appetite. Her father has been actively tuberculous with positive sputum for the last three years. Tonsillectomy done two years before admission was without any apparent beneficial results. The physical examination showed an underweight of 5 per cent, a strongly positive tuberculin reaction, anterior and posterior cervical gland and submaxillary glands, plus 3 and plus 4. The chest was normal. The antero-posterior roentgenogram (fig. 4) showed a large granular-appearing mass, *A-B*, overlying the right auricle opposite the seventh rib. On first reading, shortly after admission, this shadow was entirely overlooked. The oblique film (fig. 5),

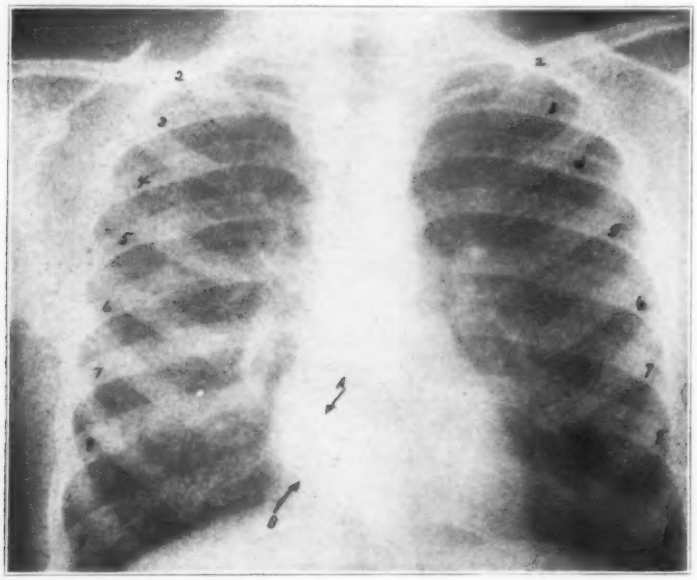

Fig. 4 (case 2).—A calcium-bearing gland *A-B*, overlying the right auricle.

which is a part of a stereo-roentgenogram, showed the mass *A-B* in the posterior mediastinum, overlying the thoracic vertebra. This shadow is interpreted as due to a semicalcified caseous lymph node, the exact anatomic location of which cannot be determined; hence the term intrathoracic glandular tuberculosis. The symptoms are probably due to this active focus.

During the six months of residence in the preventorium, the usual daily range in temperature was from 98 to 100.5 F. After complete rest for one month, the temperature subsided to normal, only to recur on the resumption of physical activity. The child is, however, otherwise playful and has gained 4 pounds (1,814.3 Gm.). This patient is still under observation.

CASE 3.—*Intimate Exposure, Definite Symptoms, Misleading Physical Signs, Roentgen-Ray Diagnosis of Tracheobronchial Tuberculosis.*

S. B., a boy, aged 10, was admitted to the preventorium on July 13, 1927, giving a history of frequent colds, persistent dry cough, intermittent fever and weakness for more than a year. The father had had active tuberculosis for many years.

Tonsillectomy was performed two years before admission. Physical examination showed: underweight, 12 per cent; tuberclin reaction, strongly positive; anterior cervical glands on right, plus 3, and only barely palpable on the left. A loud systolic murmur was heard at the base of the heart, pronounced over the pulmonic area but also distinctly audible over the aortic area. The lungs were normal. The roentgenogram showed fairly normal hila and clear pulmonic fields, but opposite the aortic arch and near the sixth costovertebral junction on the right side (fig. 6 *A-B*) could be seen an olive-shaped lymph gland which, in all probability, was in a state of partial caseation. The pressure of the gland on the large vessels may be responsible for the murmurs heard at the base of the heart.

The clinical course, during the first four months, was active, characterized by intermittent fever up to 100.5 F., general indisposition and spasmodic cough. Under rest and heliotherapy, the symptoms subsided. Normal weight had been

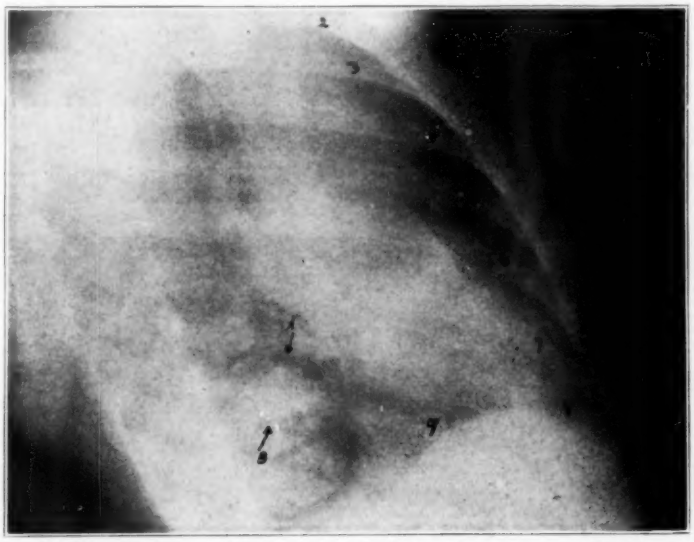

Fig. 5 (case 2).—Oblique view, showing more clearly the semicalcified caseous lymph node *A-B*.

attained at the time of discharge. A follow-up roentgenogram did not show evidence of any recession in the glandular enlargement. Observation was continued in the outpatient department.

CASE 4.—*Intimate Exposure, Disease of the Hip and Tracheobronchial Tuberculosis with Apparent Recovery, Followed by Miliary Tuberculosis and Death Three Years After Discharge.*

P. M., a girl, aged 12 years, was admitted to the preventorium on May 6, 1921, on account of intermittent fever, weakness, slight pain in the right hip and limping in the morning. The father died five years before admission of active pulmonary tuberculosis. Physical examination showed: underweight, 18 per cent; chest, normal; slight muscular spasm over the right hip and pain on motion of the joint. The roentgenograms showed early changes in the bones of the head of the femur. The roentgenogram of the chest showed a cloudy hilum on the right, with increased vascularity of the lower trunk. On the left side between the fifth and 6th ribs (fig. 7 *A-B*) can be seen semicalcified lymph glands.

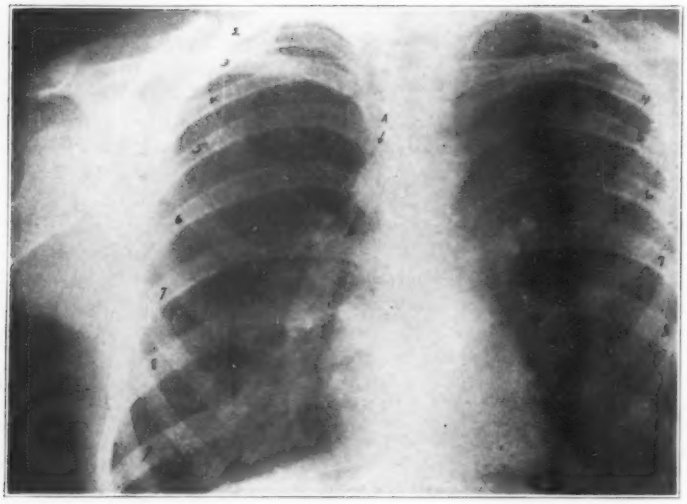

Fig. 6 (case 3).—An olive-shaped tuberculous lymph node, *A-B*, causing pressure on the large vessels and giving rise to murmurs at the base of the heart.

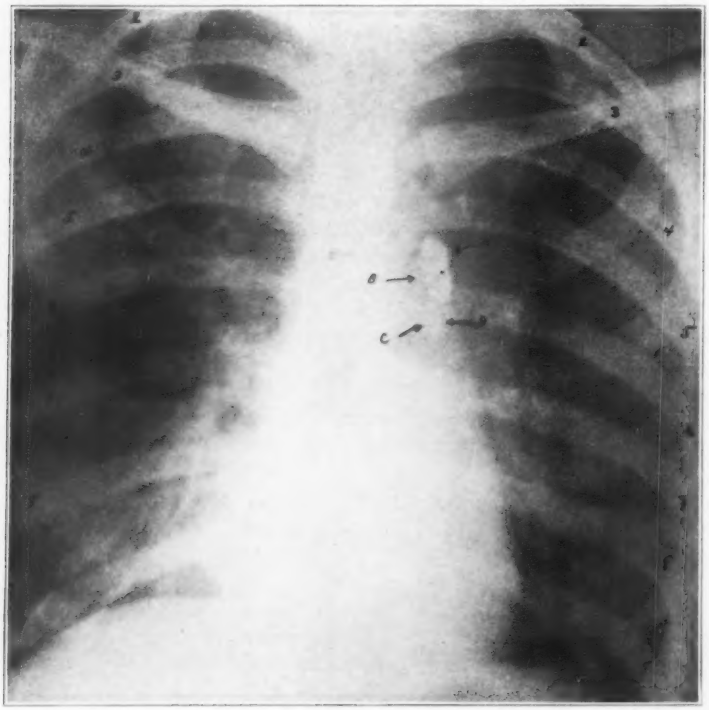

Fig. 7 (case 4).—An abnormal right hilum and calcified glands *A-B* and *C-D* on the left side. Miliary tuberculosis and death three years from date of apparent recovery.

Under immobilization and heliotherapy, marked local and general improvement was noted. At the time of discharge in September, 1925, the child seemed fully recovered, although the affected extremity needed some continued mechanical support. She remained at home doing housework.

The subsequent history and clinical course were furnished by Dr. F. R. Trout of Denver, who advised that he was first called in August, 1927, on account of "cessation of menstruation, headache and a cold in the chest." At this time, Dr. Trout found many moist râles over the lower part of the left lung. Owing to a high temperature of 103 F., he at first made a diagnosis of lobar pneumonia. The course continued to be active. Excessive cough and expectoration were the leading symptoms. The sputum was negative. In October, abdominal cramps and diarrhea became manifest and persisted until the end. On Dec. 15, 1927, tubercle

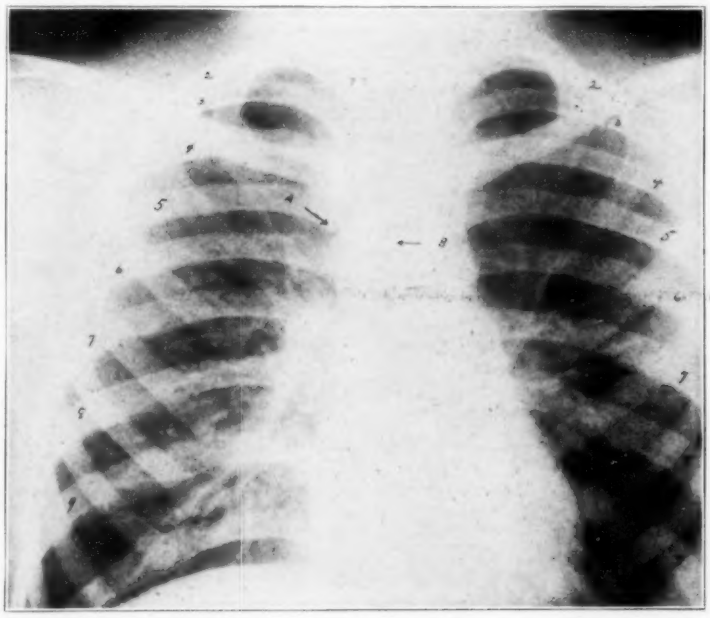

Fig. 8 (case 5).—The suspicious shadow *A-B* should be noted. On first reading, this roentgenogram was interpreted as normal.

bacilli were recovered from the sputum. At this time there were scattered moist râles all over the chest and marked tenderness over the abdomen. Death occurred in December, 1927. The clinical course, in Dr. Trout's opinion, was characteristic of miliary tuberculosis. Unfortunately, a roentgenogram of the chest was not obtained.

CASE 5.—*Intimate Exposure, Persistent Low-Grade Fever, Misleading Physical Signs.*

G. F., a boy, aged 10 years, was admitted to the preventorium on Feb. 15, 1927, on account of fever and underweight. His father was a patient at the hospital and is still attending the outpatient department. Physical examination showed: underweight, 16 per cent; tuberculin reaction, strongly positive; posterior cervical lymph nodes on the right, plus 2 and on the left, plus 3. The tonsils were small

and submerged but apparently not diseased. The chest was essentially normal, except for a soft blowing systolic murmur at the apex transmitted to the axilla. The roentgenogram was apparently negative.

After several weeks of rest, the temperature became normal, only to recur following physical exertion. On a few occasions, some dyspnea and scattered sibilant râles were noted. These symptoms, associated with a murmur in the mitral area, aroused attention to the heart, but neither physical, roentgenographic or electrocardiographic examinations showed any evidence of cardiac decompensation. Because of an ill defined shadow overlying the spine on the right side near the fifth and sixth ribs, as noted on the anteroposterior film (fig. 8 *A-B*), it was decided to take another exposure with the patient in the oblique position.

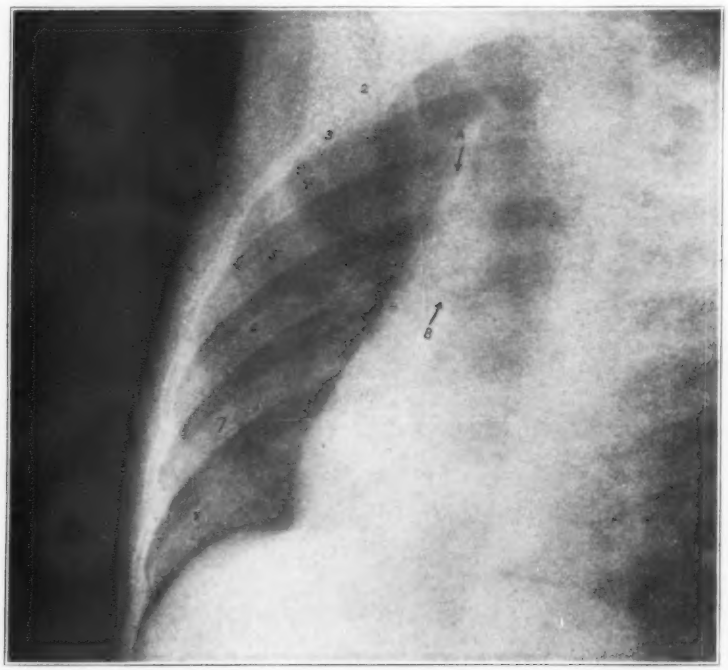

Fig. 9 (case 5).—Oblique view, showing a granular appearing lymph node in a probable state of caseation necrosis, *A-B*.

Figure 9 *A-B* shows a large granular oval-shaped lymph node, which is considered the active focus responsible for the symptoms. This patient is still under treatment.

CASE 6.—*Intimate Exposure, Pulmonary Tuberculosis, Negative Physical Signs, Recovery.*

A. K., a girl, aged 11 years, was admitted to the preventorium on Sept. 23, 1920, on account of loss of weight and intermittent fever. As her father died of pulmonary tuberculosis a year before admission, the mother was apprehensive and anxious to place the child in the preventorium for observation. Physical examination showed: Pirquet test, positive; cervical lymph nodes not palpable; tonsils not diseased. The chest was normal. The child was 5 per cent underweight.

The roentgenogram of the chest (fig. 10) disclosed a discrete fibrous spot at each apex (*A-B* and *C-D*); infraclavicular infiltration on the right side, apparently extending from the hilum along the upper trunk (*E-F*); a widened right hilum with discrete calcium-bearing glands hugging the border of the right auricle (*G-H*); a probable primary focus at the leaf of the right diaphragm (*I-J*), and a large granular circular lymph node, nearly the size of a large olive, at the left hilum (*K-L*).

During the first two months of complete immobilization, the patient had four febrile reactions, the temperature mounting to 100 or 101 F., lasting from twenty-four to forty-eight hours. There were no other complaints. Figure 11 shows the

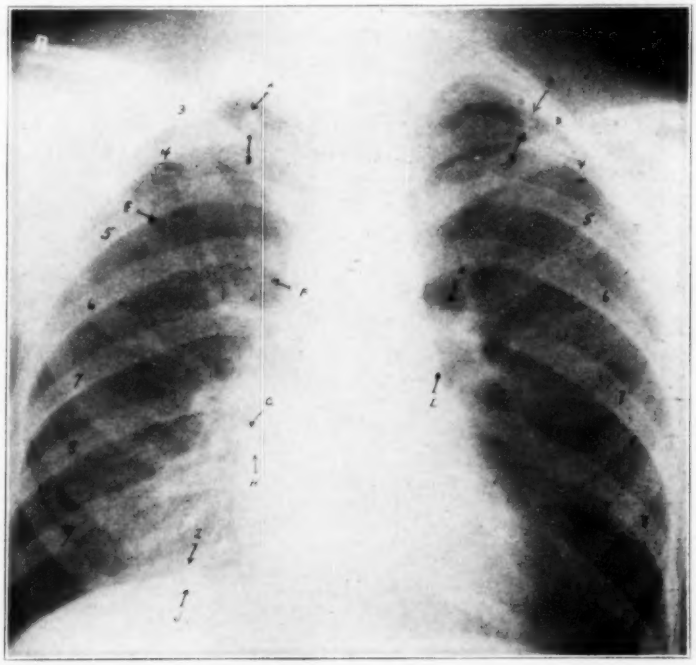

Fig. 10 (case 6).—Tracheobronchial and pulmonary tuberculosis in a child, aged 11 years. Discrete fibrous spot at each apex (*A-B* and *C-D*); infraclavicular infiltration on right (*E-F*); widened right hilum with discrete calcium-bearing glands hugging the border of the right auricle (*G-H*); probable primary focus at leaf of right diaphragm (I-J); large granular circular lymph node at left hilum (*K-L*).

temperature record, which is fairly typical of many of the tracheobronchial cases in which pulmonary involvement was not present. After another month of rest, during which the temperature remained normal, the child was put on graduated exercise and routine treatment. The fever did not recur. She was discharged in October, 1921, in an excellent state of health with an overweight of 4 pounds (1,814 Gm.). Thus far, it has been impossible to trace her present whereabouts.

It is not within the scope of this paper to discuss frank pulmonary tuberculosis in children and its clinical course. This subject is reserved for another publication after complete follow-up data have become available. From the records and roentgenograms that are now available, it appears that the prognosis is not as unfavorable as is generally supposed, for quite a number of patients with active apical or infraclavicular infiltration pursued a benign course and under treatment made apparently satisfactory recoveries.

SUMMARY

Clinical records of 300 preventorium children were reviewed and a study made of the roentgenograms. An underweight of 7 per cent or

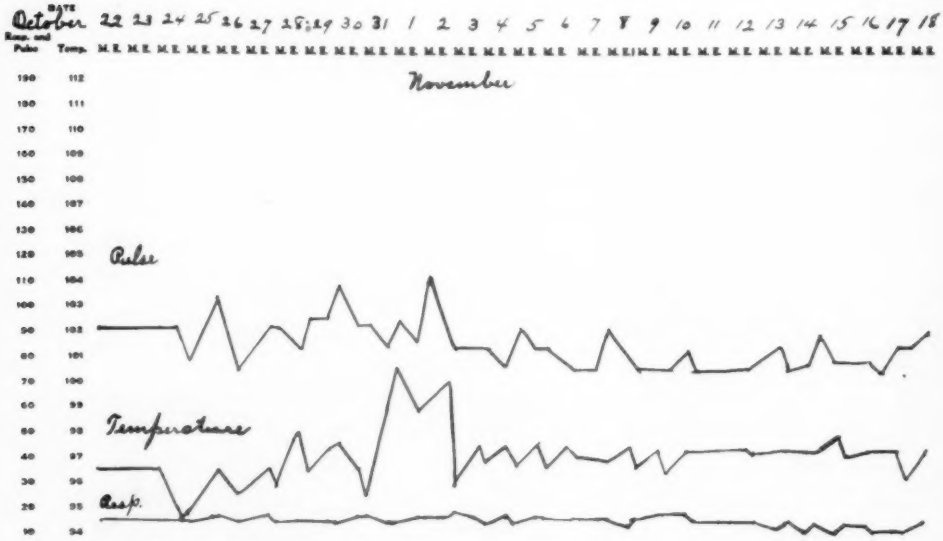

Fig. 11 (case 6).—Graphic record, which is typical of other cases showing active tracheobronchial tuberculosis.

more was found in 70 per cent. In 45 per cent definite cervical or submaxillary lymphadenitis was noted. In 30 per cent, fever was a prominent symptom. An appreciable difference was not observed in the symptomatology or physical signs between the tonsillectomized and the nontonsillectomized groups. The greatest incidence of clinical disease and positive physical signs was noted in those with a history of definite exposure. Physical signs were often negative when the history and symptoms pointed to active disease. The x-ray was of the greatest aid in diagnosis when correlated with the other symptoms. Of the entire series, only seventy-two roentgenograms of the chest were considered within the normal. Calcification at the hilum, strongly suggestive of tuberculosis, was observed in only 22 per cent, but it is believed that the

incidence would have been greater if films in the oblique position had been taken. Primary foci were detected in 11.6 per cent, and in 11.3 per cent was noted frank parenchymal pulmonary infiltration. Attention is called to the difficulty of making a diagnosis of tracheobronchial tuberculosis, which in this series was frequently responsible for active symptoms and was often attributed to other causes. Under treatment, symptomatic improvement invariably occurred. Unremitting supervision was necessary, for in many patients there was a recurrence of symptoms with increased physical activity. A notable recession was not noted in either the cervical or the tracheobronchial glands after prolonged periods of continued heliotherapy.

CONCLUSIONS

1. Active juvenile tuberculosis is still unrecognized in the majority of instances.

2. Physical signs of juvenile tuberculosis are indefinite and often misleading.

3. The history and symptoms are more important as a basis for treatment than physical signs.

4. The x-ray is of the greatest aid in diagnosis.

5. The practical solution of the problem of tuberculosis in adults depends in a large measure on the attention that is accorded children with latent or active tuberculosis.

6. All patients who have been exposed to the disease should have a limited period of residence in a suitable hospital or should be under continued close medical supervision even in the absence of symptoms.

7. Preventoriums for the observation of children who have been exposed and the treatment for juvenile tuberculosis are needed as urgently as sanatoriums for the treatment of adults with tuberculosis.

A COMPARISON OF COD LIVER OIL AND ULTRAVIOLET LIGHT FOR USE IN THE PREVENTION OF RICKETS*

ARTHUR D. HOLMES, PH.D.

EDWIN T. WYMAN, M.D.

LAWRENCE W. SMITH, M.D.

AND

MADELEINE G. PIGOTT, B.S.

BOSTON

Numerous investigators have demonstrated that cod liver oil is the richest available natural source of fat-soluble vitamins. The antirachitic vitamin content of cod liver oil prepared and stored under proper conditions justifies its being considered as specific for rickets. More recently, investigators have demonstrated that ultraviolet light has definite therapeutic value in either the prophylactic or the curative treatment of patients with rickets. This is true of both the natural ultraviolet light present in sunshine and the ultraviolet light produced artificially by the mercury vapor quartz lamp and by some other sources of artificial light. As cod liver oil and ultraviolet light have been proved to be reliable agents for treatment in cases of rickets, the pediatrician naturally is interested to have information concerning the relative efficacy of cod liver oil and ultraviolet light as a source of antirachitic activity. This investigation was undertaken to obtain data which would shed some light on this question.

NATURE OF COD LIVER OIL USED

Cod liver oil used in the experiments reported here was prepared by the direct steam method from livers taken from fish caught by shore fishermen along the coast of Massachusetts and by steam trawlers operating out of Boston and New York. Two thirds of the livers were obtained from the shore fish and one third from fish taken by steam trawlers. The crude cod liver oil obtained by the steam method was chilled by a continuous brine cooling system. A plate and frame press was used to separate the nonfreezing oil from the stearin. The chemical and physical characteristics of the pressed oil were determined by the methods described in the Pharmacopeia of the United States.[1] The results of the analysis are seen in table 1.

* Received for publication, July 5, 1928.

1. The Pharmacopeia of the United States, ed. 10, Philadelphia, J. P. Lippincott Company, p. 263.

When these results are compared with the specifications for cod liver oil outlined in the Pharmacopeia, it will be noted that each of these values is in accordance with the official specifications. Accordingly, one would conclude that the chemical and physical characteristics of the oil under investigation were typical of those of average medicinal cod liver oil. The vitamin A content of the oil under consideration was determined by the method described [2] in the Pharmacopeia, and it was found that this oil had a vitamin content of 1,250 units per gram.

SOURCE OF ULTRAVIOLET LIGHT

For the purpose of this study, ultraviolet light was produced by a mercury quartz Cooper-Hewitt lamp operated with a 110 volt alternating current obtained from the city lighting circuit. A voltmeter and ammeter were used to measure the current across the arc. From these the wattage was computed, and this is shown in figure 1. After the lamp had been run long enough so that ammeter and voltmeter readings were fairly constant, the ultraviolet light was used for fifteen minute

TABLE 1.—*Chemical and Physical Characteristics of Cod Liver Oil*

Specific Gravity	Index of Refraction	Saponification Value	Iodine Number	Free Fatty Acids	Unsaponified Matter
0.922	1.4782	183.8	163.6	0.6%	1.32%

periods. Four voltmeter and ammeter readings were made for each experimental period, one at the beginning of the period and the others after five, ten and fifteen minutes, respectively. The ammeter and voltmeter readings were averaged, and the product is reported as the daily wattage in the curves appearing in figure 1. The wattage varied materially from day to day, obviously owing to fluctuating conditions in the city lighting circuit (fig. 1). Unfortunately, we do not know whether this variation of intensity of current is greater or less than that of current used by other investigators conducting studies of ultraviolet light. In fact, we are unable to judge as to whether the intensity of the ultraviolet light used in these tests is at all similar to that of ultraviolet light used by other investigators, for those who have studied the antirachitic activity of ultraviolet light have not ordinarily supplied data concerning the intensity of the ultraviolet light used.

METHOD OF IRRADIATION

In the experiments described here, the animals were irradiated for fifteen minutes daily at a distance of 36 inches (90 cm.). A special cage, which held four animals, was devised for this purpose. It was so constructed that each animal

2. The Pharmacopeia of the United States, ed. 10, p. 469.

was in a separate compartment, and thus could not shadow or be shadowed by another animal (fig. 2). Since the intensity of ultraviolet radiation varies inversely with the square of the distance of the animal from the source of ultraviolet light, it obviously follows that the experimental animals should be kept at a constant distance from the source of ultraviolet light rather than that the animals should be permitted to vary the intensity of ultraviolet radiation by moving from time to time from the top to the bottom of cages the height of which is a relatively

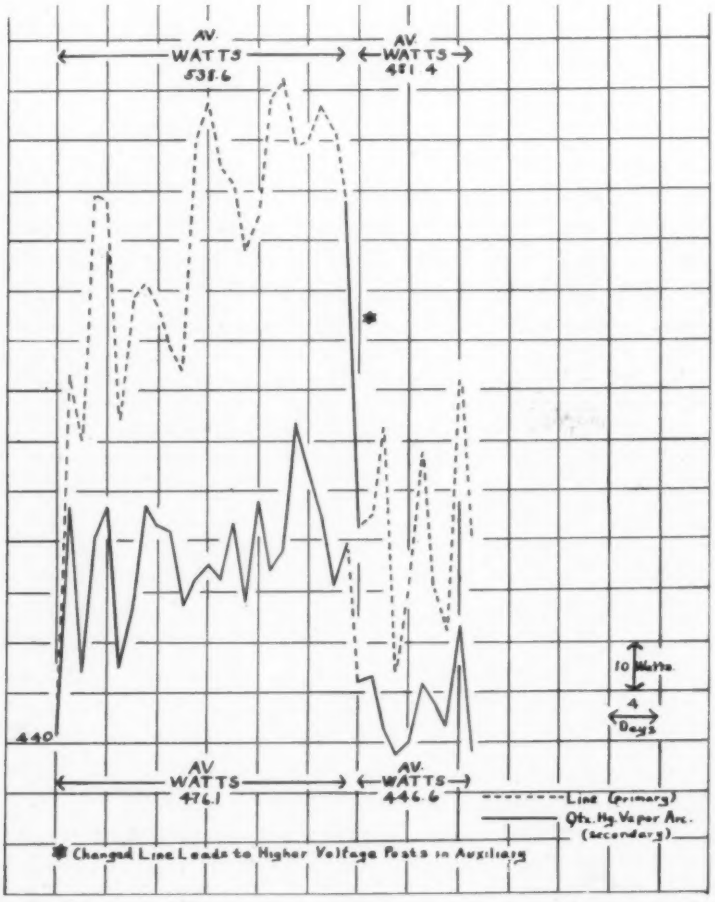

Fig. 1.—Curve representing daily variation in wattage of the Cooper-Hewitt quartz mercury vapor arc.

large fraction of the distance from the cage to the source of ultraviolet light. The cages illustrated in figure 2 are 2 inches (5 cm.) in height and thus keep the animals at a constant distance from the lamp supplying the ultraviolet light. The cages were arranged about a hexagon, and the center of the upper surface of the cage was 36 inches from the center of the quartz tube which emitted the ultraviolet radiation. To eliminate shadows, the cages were tipped so that the upper surface of the cage was perpendicular to the line from the cage to the lamp.

As the different cages occupied slightly different positions in relation to the quartz tube containing the mercury vapor, the cages were always placed in the same order and each day were moved progressively around the hexagon until at the end of the experiment each cage had occupied each position six different days. Hence the animals comprising the groups receiving the direct irradiation and the direct irradiation plus 14.8 mg. of cod liver oil received comparable irradiation.

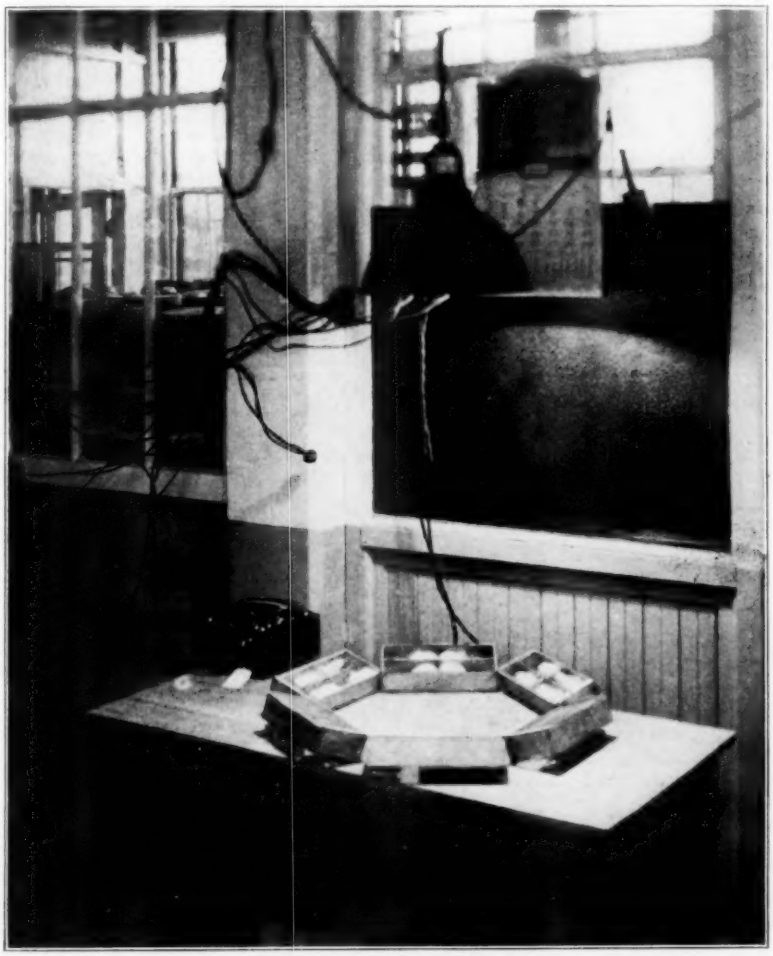

Fig. 2.—Cages arranged in hexagon in which irradiation of rats was carried out.

LABORATORY PROCEDURE

The experimental animals used in this investigation were recently weaned albino rats. They were divided into five groups which were as nearly identical as possible in regard to weight and physical conditions. The groups were designated as "controls"; "direct irradiation"; "direct irradiation plus 14.8," "4.9 mg. C.L.O.," "14.8 mg. C.L.O.," "19.8 mg. C.L.O." and "29.7 mg. C.L.O.," respectively. The

animals were placed in individual cages and were continuously supplied with food and water. Their diet was the Steenbock diet, consisting of: 76 per cent of yellow corn (ground), 20 per cent of wheat gluten, 3 per cent of calcium carbonate, and 1 per cent of sodium chloride. Each day the animals received a 0.2 Gm. yeast tablet as a supplementary source of vitamin B. The animals were kept in a colony room with a west exposure which received natural light through ordinary glass windows.

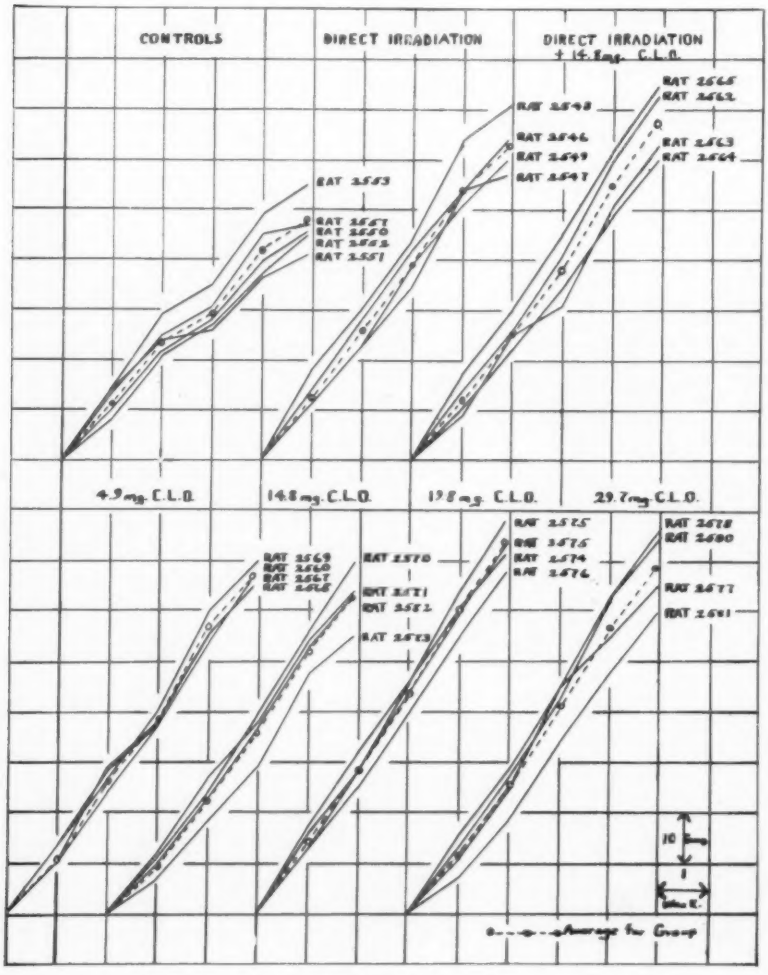

Fig. 3.—Growth records.

In this instance, the relative antirachitic activity of cod liver oil and ultraviolet light was determined on a protective basis. The experimental period was of thirty-six days' duration. A record was made of the increase in body weight. At the termination of the experiment, the rats were killed; the blood was examined for its calcium and phosphorus content; roentgenograms were made of the right femur and tibia. These were subsequently examined by a pathologist. The left

femur and tibia were carefully freed of all tissue, extracted sixteen hours with alcohol, subsequently extracted twenty-four hours with ether, dried for one hour in an electric oven at from 98 to 100 F., weighed and ashed. The results of these various examinations are discussed.

INCREASE IN BODY WEIGHT

The animals were weighed every Monday, Wednesday and Friday, and the weight of the individual animals of each group is reported in figure 3. Also, curves are included which give the computed average weight of the animals comprising each of the five groups. Figure 3 shows that the increase in body weight was materially less for the control group than for any of the other groups. It also showed that the average increase in body weight for the different groups was nearly the same for the animals receiving cod liver oil or ultraviolet light or both. The conclusion to be drawn from these results is that the administration of 5 mg. daily of the oil in question produces nearly

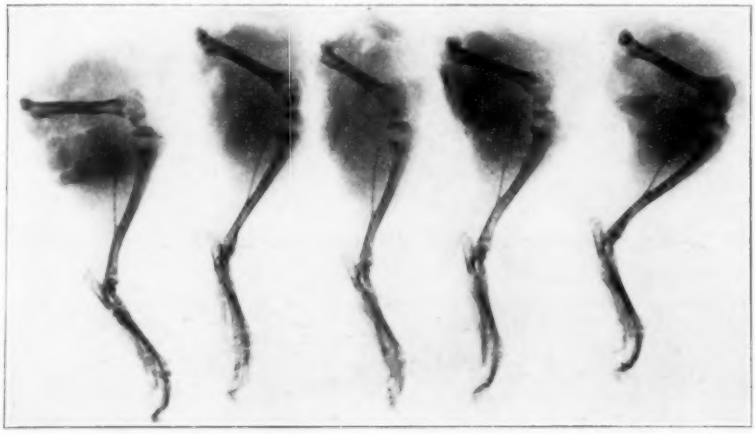

Fig. 4.—Legs of control group, with incidence of delayed calcification of femur and tibia.

the same increase in body weight as 15, 20 or 30 mg. of the same oil when used for the prevention of rickets in experimental animals fed a diet which consistently produces rickets in animals that do not receive any other food. The increase in body weight of the animals receiving fifteen minute daily irradiation of ultraviolet light as described was approximately the same as that of the animals receiving 5 mg. or more of cod liver oil daily. Also, it will be noted that the group of experimental animals which received fifteen minute irradiation with ultraviolet light and 15 mg. of cod liver oil grew slightly more than the animals which received either ultraviolet light or cod liver oil alone.

ROENTGENOGRAMS

As already noted, at the end of the thirty-six day experimental period, roentgenograms were made of the right leg of the experimental animals comprising the seven different groups under consideration (figs. 4 to 10 inclusive). Figure 4, which is the roentgenogram of the control group, furnishes sufficient evidence of

delayed calcification of both femur and tibia to indicate that these animals had rickets. In general, the extent of calcification of the femur and tibia of the remaining six groups, i. e., direct irradiation (fig. 5); direct irradiation plus 14.8 mg. of cod liver oil (fig. 6); plus 4.9 mg. of cod liver oil (fig. 7); plus

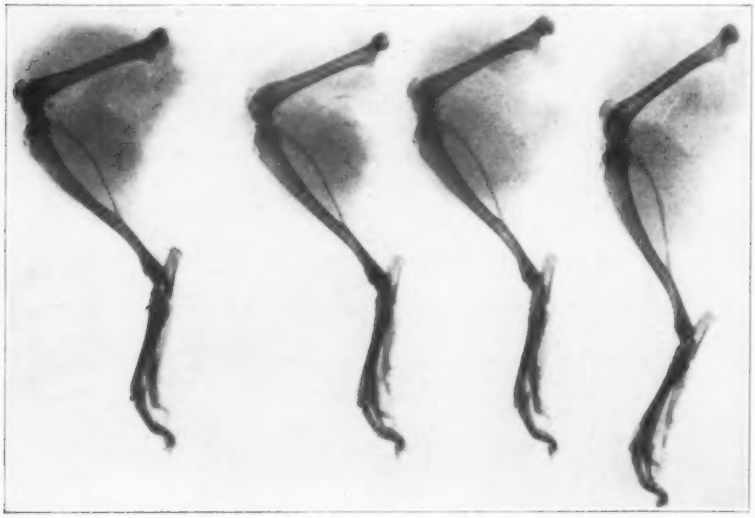

Fig. 5.—Legs of group receiving direct irradiation with ultraviolet light.

Fig. 6.—Legs of group receiving direct irradiation plus 14.8 mg. of cod liver oil.

14.8 mg. of cod liver oil (fig. 8); plus 19.8 mg. of cod liver oil (fig. 9) and plus 29.7 mg. of cod liver oil (fig. 10), is nearly, if not actually, the same. Accordingly, these results would indicate that 5 mg. of the oil under consideration is sufficient for preventing the development of rickets in rats maintained under the experimental conditions employed in this study.

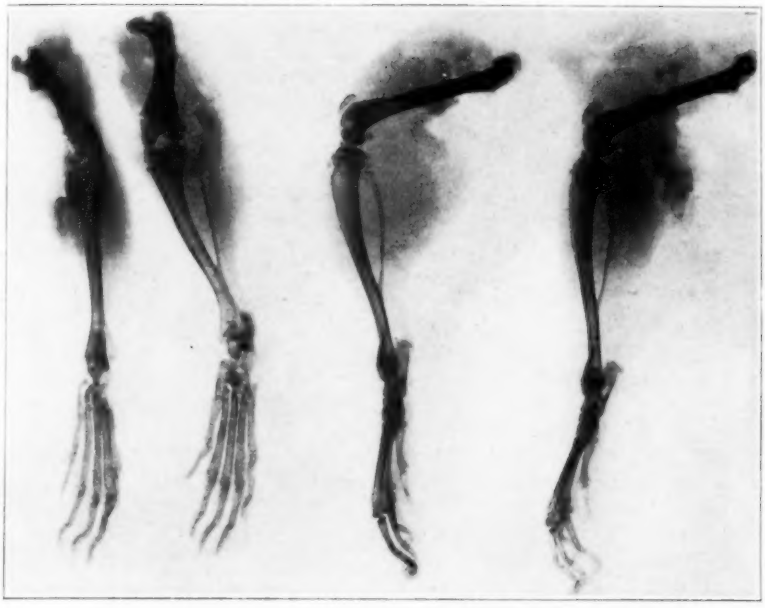

Fig. 7.—Legs of group receiving 4.9 mg. of cod liver oil.

Fig. 8.—Legs of group receiving 14.8 mg. of cod liver oil.

UNIV. OF MICH.

Fig. 9.—Legs of group receiving 19.8 mg. of cod liver oil.

Fig. 10.—Legs of group receiving 29.7 mg. of cod liver oil.

PATHOLOGIC EXAMINATION OF FEMUR AND TIBIA

As in previous investigations, the histologic examination of the tissues was made in as uniform a fashion as possible, identical sections being cut through the femur and tibia of representative animals in each group. In order to simplify the tabulation of the results, an attempt was made to grade the degree of rachitic change or deformity. Four gradations were arbitrarily established as follows:

Grade 1.—Normal—The normal rachitic change is a narrow, straight line of ossification at the epiphysis. The cartilage is uniform in its staining reaction and its cells are regular in size. The transition from cartilage to bone is evidenced by the gradual laying down of osseomucin rather than by irregular calcification, and this appears in a limited zone which is symmetrically distributed across the section. The bone marrow merges almost abruptly in this area with the stroma of the proliferating cartilage.

Grade 4.—Advanced Rickets—Advanced rickets is represented by the group of animals on a control diet designed to produce marked rickets within twenty-eight days. The epiphyseal junction shows profound changes. The line of ossification is extremely thickened, but the density of the tissue is markedly reduced as the result of some impairment of function of the cells to lay down osseomucin. The picture is that of osteoid tissue rather than true bone. The inherent lesion seems to be associated with a derangement of the cartilage cells. They are increased in size and number. There are many nests of large embryonic-appearing cartilage cells, the stroma and intercellular matrix of which appear to be inexplicably confused, in places approaching a myxomatous state. There is a gradual replacement of the marrow in this region by connective tissue and newly formed blood vessels.

Grades 2 and 3.—These grades represent stages between the two extremes. There is some opportunity for the personal equation to enter into a limited extent, just as it may in the interpretation of the various stages of development of the myeloblast through the premyelocytic, the myelocytic and the metamyelocytic phases to the adult leukocyte. By establishing only four gradations, however, one can fairly readily recognize the lesion as being normal, early, moderate or advanced in type, which is about as far as any classification warrants our procedure.

On this basis, then, the animals in each group were examined and graded histologically.

The animals were placed in seven groups. Of these, the first group represented the control animals on a diet producing experimental rickets within from three to five weeks. These animals all showed grade 4 changes in their long bones.

The second group was made up of animals on the same diet, plus irradiation by the ultraviolet lamp, without the addition of cod liver oil. These animals at the end of the experimental period showed barely appreciable histologic changes in the long bones; on the basis of the gradation cited, they were placed in grade 2.

The third group was composed of animals on the standard rachitic-producing diet plus 14 mg. of cod liver oil daily, and the same direct irradiation was given as in the preceding group; they showed practically no demonstrable rickets by the histologic preparations. These were accordingly grouped as grade 1.

The fourth group received the standard control diet plus 4.9 mg. of cod liver oil daily without irradiation, either of the animals or of the oil. At the end of the five weeks' experimental period, these animals histologically showed definite but mild degree of rachitic changes in the epiphyses of the long bones, and were placed in grade 3.

The fifth, sixth and seventh groups were treated comparably, receiving, respectively, 14.8, 19.8 and 29.7 mg. of cod liver oil daily, together with the standard rachitic-producing diet. At the end of the experimental period of thirty-six days, group five showed demonstrable changes sufficient to include them in the histologic classification of grade 2. The sixth and seventh groups histologically did not show any more variation than is found in the ordinary control group. Individual animals might be classed in grade 2, but the groups as a whole were essentially nonrachitic.

It is obviously difficult to quantitate absolutely or to classify as sharply as we should like the slight changes which were seen in a relatively small number of experimental animals over such a short period of time. To obviate any preconceived opinion, the animals were recorded simply by numbers, and the pathologist at the time of classifying the lesions was unaware of the experimental group to which they belonged. Only in groups 5, 6 and 7 were any discrepancies noted, and these were merely a question as to whether they belonged in grade 1 or 2.

When the slides are reviewed it is easy to see how one can be misled into trying to make more accurate differentiation between the various groups. In general, it is our impression that the animals receiving over 15 mg. of the oil showed rather less tendency to demonstrable histologic change than the animals which were simply irradiated. No appreciable difference was noted in the animals receiving an adequate amount of cod liver oil plus irradiation.

CALCIUM AND PHOSPHORUS OF BLOOD

The blood obtained from the individual animals of the different groups was pooled and centrifugalized. The blood serum thus obtained was analyzed for calcium and phosphorus, and the results obtained are shown in table 2.

Reference to the calcium and phosphorus content of the blood serum of the different groups will show that both calcium and phosphorus were materially lower for the controls than for any of the remaining groups. The calcium content of the blood serum of the groups other than the controls showed a variation of less than 2 per cent, namely, 12.8 per cent for the group receiving direct irradiation plus 14.8 mg. of cod liver oil, and 11.1 per cent for the group receiving 4.9 mg. of cod liver oil.

The phosphorus content of the blood serum of the groups other than the controls showed a 2.2 per cent variation, the largest being 7.6 per cent for the groups receiving 4.9 mg. of cod liver oil and 19.8 mg. of cod liver oil, and the smallest, 5.4 per cent, for the group receiving direct irradiation.

The product of the calcium and phosphorus content of the blood serum of the controls was decidedly lower than that for the remaining groups. While the product of calcium and phosphorus for the group receiving

TABLE 2.—*Calcium and Phosphorus of Blood Serum*

			Direct Irradiation				
	Controls Per Cent	Direct Irradiation Per Cent	+ 14.8 Mg. of Cod Liver Oil Per Cent	4.9 Mg. of Cod Liver Oil Per Cent	14.8 Mg. of Cod Liver Oil Per Cent	19.7 Mg. of Cod Liver Oil Per Cent	29.7 Mg. of Cod Liver Oil Per Cent
Calcium	10.2	11.8	12.8	11.1	12.2	11.2	12.3
Phosphorus	3.7	5.4	7.0	7.6	6.9	7.6	7.3
Product of calcium and phosphorus ..	37.7	63.7	89.6	84.4	84.2	85.1	89.8

direct irradiation was decidedly higher than for the controls, it was nevertheless materially lower than for the remaining five groups. The values for the groups of animals receiving 4.9, 14.8 and 19.8 mg. of cod liver oil daily were practically the same; also those for the groups and 14.8 mg. of cod liver oil daily. It would therefore appear from the of the results of the analyses of the blood, it appears that 5 mg. of the cod liver oil under consideration possessed a materially higher antirachitic value than direct irradiation with ultraviolet light for fifteen minutes daily at a distance of 36 inches.

ASH CONTENT OF FEMUR AND TIBIA

The ash content of the left femur and tibia, obtained as described, is reported in table 3, separately for the femur, for the tibia, and as a computed average of both the femur and tibia.

It will be noted that the ash content of the tibia was consistently higher than that of the femur. This is in agreement with the difference between the ash content of the tibia and of the femur obtained in a study of the value of different types of glass for transmitting ultra-

violet light.[3] The marked difference between the ash results obtained with the controls and the ash results of the remaining groups gives striking evidence of the antirachitic value of cod liver oil and ultraviolet light. The ash content of both the femur and the tibia was slightly greater for the group fed 14.8 mg. of cod liver oil than for the group fed 4.9 mg. Also it was slightly greater than that of the groups fed 14.8 mg. of cod liver oil.

of the group treated with direct irradiation was nearly equivalent to that of the group fed 14.8 mg. of cod liver oil and was nearly the same as the ash content of the bones of the animals receiving both irradiation and 14.8 mg. of cod liver oil daily. It would, therefore, appear from the results of the ash determinations that fifteen minute daily irradiations with the ultraviolet light used in this investigation is approximately equal in antirachitic value to that of 14.8 mg. daily of the cod liver oil under consideration.

TABLE 3.—*Ash Content of Femur and Tibia*

			Direct Irradiation				
	Controls Per Cent	Direct Irradiation Per Cent	+ 14.8 Mg. of Cod Liver Oil Per Cent	4.9 Mg. of Cod Liver Oil Per Cent	14.8 Mg. of Cod Liver Oil Per Cent	19.8 Mg. of Cod Liver Oil Per Cent	29.7 Mg. of Cod Liver Oil Per Cent
Femur	31.8	54.0	53.0	52.5	55.3	54.1	52.2
Tibia	39.7	55.1	55.4	53.6	56.4	55.4	53.3
Combined	35.7	54.6	54.2	53.0	55.8	54.7	52.7

SUMMARY

A comparison has been made of the mineral metabolism of seven groups of rats, controls, direct irradiation, and direct irradiation plus 14.8, 4.9, 14.8, 19.8 and 29.7 mg. cod liver oil, respectively, which were maintained for thirty-six days, after weaning, on a rachitic-producing diet and on the same diet supplemented with ultraviolet light or cod liver oil.

As judged by the growth, roentgenograms of the femur and tibia, calcium and phosphorus of the blood serum and mineral content of the femur and tibia, the mineral metabolism of the controls was decidedly inferior to that of the animals receiving the rachitic diet supplemented with ultraviolet light or cod liver oil. Aside from the control group, the average increase in body weight during the thirty-six day experimental period was greatest for the group fed 19.8 mg. of cod liver oil and least for the group receiving direct irradiation and the group fed 14.8 mg. of cod liver oil.

3. Wyman, E. T.; Holmes, A. D.; Smith, L. W.; Stockbarger, D. C., and Pigott, M. G.: The Value of Different Types of Glass for Transmitting Ultraviolet Light, Am. J. Dis. Child., to be published.

The roentgenograms of the right femur and tibia of the groups of animals other than the controls show little if any difference in the extent of calcification.

The calcium content of the blood serum of the groups other than the controls is in rather close agreement. The phosphorus content of the blood serum for the group receiving direct irradiation is definitely higher than that of the controls, but materially lower than that of the other five groups. The product of the calcium and phosphorus content of the blood serum is 37.7 per cent for the controls and 63.7 per cent for the group receiving direct irradiation, and for the remaining groups it is in excess of 84 per cent. The highest values, 89.6 per cent and 89.8 per cent, were for the groups receiving direct irradiation plus 14.8 mg. of cod liver oil and 29.7 mg. of cod liver oil, respectively.

In general, then, the results here reported show that 5 mg. of the cod liver oil under consideration has sufficient antirachitic activity to protect laboratory animals against rickets when maintained under the conditions described. The results also indicate that 5 mg. of the oil under consideration gives greater protection than fifteen minutes of irradiation with ultraviolet light at a distance of 36 inches, and that 5 mg. of this cod liver oil appears to be as effective as larger amounts when used as a protection against rickets.

In conclusion, attention is earnestly directed to the fact that the results herein reported apply only to the ultraviolet irradiation conditions and the cod liver oil used in this investigation, and that many additional data are required before a general comparison can be made between the antirachitic value of ultraviolet light with that of average medicinal cod liver oil.

AN INVESTIGATION OF THE PRENATAL FACTOR IN THE SUSCEPTIBILITY OF INFANTS TO RICKETS*

ALFRED F. HESS, M.D.
AND
MILDRED WEINSTOCK, B.S.
NEW YORK

It has long been recognized that there is a distinct individual susceptibility to the development of rickets—that infants brought up apparently under the same environment and given the same diet differ markedly in their tendency to develop this disorder. Formerly, clinicians were content to call attention to this phenomenon, but recently children's specialists and biochemists have attempted to harmonize this observation with the newer knowledge, and have predicated a prenatal lack of the antirachitic factor in infants who showed a predisposition to rickets and, on the other hand, a storage in those who showed no such tendency. The analogy for this hypothesis is the well known theory of Bunge in relation to anemia. It will be remembered that this physiologist ascribed the development of anemia in later infancy, and the vulnerability of certain infants in particular, to a lack of an "Eisendepot" in the liver. Similarly, in regard to rickets, there has been presupposed a greater or less store at birth of antirachitic factor in the liver—the reservoir of vitamins and nutritional factors. As far as we are aware, an investigation of the validity of this hypothesis has never been carried out, although it is accessible to experimental study.

It is well established that there is an essential difference in susceptibility to rickets between the premature and the infant born at full term. The former can only with difficulty be prevented from developing this disorder, even when nursed and given full amounts of cod liver oil. It seemed, therefore, that a direct approach to the question which we had in mind could be gained by ascertaining whether a distinct difference in concentration of antirachitic factor could be determined in the livers of these two classes of infants. If such a distinction was not found in these extreme groups, the theory of storage as a dominant factor in susceptibility could not have a real foundation. With this object in view, we have assayed biologically, during the past year, the livers of a considerable number of infants who were born and died soon after birth at the Sloane Hospital for Women and a few from the Nursery and

* Received for publication, July 24, 1928.

* From the Department of Pathology, College of Physicians and Surgeons, Columbia University.

Child's Hospital, hospitals which provided us with the necropsy material. In addition to these specimens, we have had the opportunity of testing the livers of some older infants, selected because we were conversant with the details of their diet, as well as the livers of some adults.

Accordingly, the cases were divided into two main groups—those infants who were born at about full term and others who, in view of their length and weight, must be considered as born prematurely. In this regard, we followed the classification that we employed a few years ago in a study of the carpal centers of new-born infants, and considered all infants shorter than 48 cm. in length and weighing less than 6 pounds (2,700 Gm.) as premature.

In view of the standardization of the biologic test, it hardly seems necessary to go into the details of the technic. Young rats were fed the low-phosphorus Sherman-Pappenheimer ration and a definite supplement of liver tissue was given separately in order to ascertain whether by this means healing or calcification was brought about. As a rule, 1 or 2 Gm. of liver tissue was fed daily.[1] It may be added that analyses showed that the liver contains approximately 1 mg. of phosphorus per gram of substance, so that in giving 1 Gm. a day, we added approximately 14 mg. of phosphorus per hundred grams of basal ration; the addition of 2 Gm. added 28 mg. per cent. Experiments carried out several years ago in connection with the initial study of this rickets-producing ration showed that such additions of phosphorus are insufficient to afford protection; the same is true for the healing process.

Tables 1, 2 and 3 reproduce the results of our study. Table 1 shows the effect of supplementing the ration by 1 or 2 Gm. of liver of full-term infants, and table 2 the effect of adding similar amounts of liver of premature infants. It may be noted that in practically no instance did 1 Gm. of "full-term liver" bring about any healing and that 2 Gm. of these livers led to slight healing of the epiphyses.

In regard to the activity of the livers of premature infants (table 2), it was found that 1 Gm. of liver induced slightly more healing than in the previous series of tests, and that when 2 Gm. was fed daily the result was irregular, varying from no curative action to slight or even moderate healing. One liver showed especial potency—that of a pathologic patient with an extremely large spleen. In some cases 1 Gm. as well as 2 Gm. was tested, but in general merely a single quantity was fed, owing to the small amount of tissue available; in the premature infants, this was barely adequate for one complete test. In passing, we call attention to

1. One or 2 Gm. is a considerable amount when compared to the quantity of fish liver which is necessary to insure protection or healing. For example, liver of the cod may bring about calcification when 0.1 Gm. is fed, and in one instance the liver of pollock was effective when as little as 0.001 Gm. was given as a supplement.

TABLE 1.—*Antirachitic Potency of Livers of Full-Term Infants at Birth*

Weight of Rat Gm.	Amount of Liver Fed Gm.	Infant			Healing of Epiphyses (Rat)
		Weight Gm.	Length Cm.	Liver Gm.	
42-60	1	3,800	51	175	None
60-70	..	.....	..	...	None
60-66	..	.....	..	...	None
44-54	..	2,735	51	120	None
40-40	..	.....	..	...	None
46-40	..	.....	..	...	None
44-52	..	3,390	51	60	None
40-60	..	.....	..	...	None
50-40	..	.....	..	...	None (?)
42-40	..	3,790	55	205	Trace
60-50	..	.....	..	...	None
50-44	..	.....	..	...	None
50-70	2	3,800	51	175	Moderate
50-60	..	.....	..	...	Very faint trace
60-90	..	.....	..	...	None
60-70	..	.....	..	...	None
50-64	..	.....	..	...	Slight
93-90	..	.....	..	...	Very slight
30-36	..	3,160*	49	150	Very slight
40-34	..	.....	..	...	Trace (?)
40-44	..	3,315	52	130	Faint trace
42-46	..	.....	..	...	Trace
40-40	..	.....	..	...	Slight

* Large spleen.

TABLE 2.—*Antirachitic Potency of Livers of Premature Infants at Birth*

Weight of Rat Gm.	Amount of Liver Fed Gm.	Infant			Healing of Epiphyses (Rat)	Remarks
		Weight Gm.	Length Cm.	Liver Gm.		
40-44	1	1,835	40	67.4	None	
50-50	..	.....	....	.....	None	
58-64	..	1,385	41	51.8	None	
40-40	..	.....	....	.....	Faint trace	
46-45	..	.....	....	.....	Very faint trace	
50-52	..	1,350	42	29	None	
56-74	..	.....	....	.....	None	
50-60	..	.....	....	.....	None	
50-60	..	1,745	44.5	90	None	
54-60	..	.....	....	.....	None	
60-60	..	2,140	46.5	150	Trace (?)	Extremely large spleen
60-70	..	.....	....	.....	Slight	
50-60	..	.....	....	.....	Moderate	
46-49	..	280	27	12	Very slight	
52-64	2	1,360	40	67.8	None	
42-64	..	1,450	40	42	None	
66-70	..	.....	....	.....	None	
60-62	..	.....	....	.....	None	
50-50	..	.....	....	.....	None	
46-50	..	2,365	46.5	120	None	Macerated fetus
46-54	..	.....	....	.....	Slight	
50-44	..	.....	....	.....	Trace	
40-44	..	1,355	38.5	73	None	
36-30	..	.....	....	.....	Slight or moderate	
40-40	..	1,000	37	41	None	
36-40	..	1,745	36	40	None	
60-92	..	1,130	39	58	Slight	Twins
60-86	..	1,235	40.5	59	Slight	
60-64	..	.....	....	.....	Trace (?)	
44-40	..	1,745	44.5	90	None	
68-110	..	2,140	46.5	150	Trace	
60-80	..	.....	....	.....	Slight	
70-90	..	.....	....	.....	Marked	
48-54	..	.....	....	.....	Trace(?)	

the unaccountable variation in the weights of the livers, which did not parallel either the weights or the lengths of the infants. For example, a full-term infant weighing 3,390 Gm. with a liver of 60 Gm., and another infant weighing 2,735 Gm., and of equal length, with a liver of 120 Gm., are found. The same incongruity was noted in regard to the weights and measurements of the premature infants.

TABLE 3.—*Antirachitic Potency of Livers of Older Infants and of Adults*

Weight of Rat Gm.	Amount of Liver Fed Gm.	Age	Previous History	Healing of Epiphyses (Rat)	Remarks
60–70	0.5	5 months	Breast fed for ten weeks, then cereal and milk; no cod liver oil	None	Weighed 8 pounds, 14 ounces (3.9 Kg.) at birth; 19 pounds (8.6 Kg.) at time of death, which was due to pneumonia
40–50				Very faint trace	
30–40	1.0			Slight	
34–40				Slight	
40–42				Moderate	
30–40	2.0			Slight	
30–40				Marked	
38–40				Marked	
50–50	0.5	11½ months	Diet of milk, cereal and vegetables; cod liver oil, 15 Gm. daily for almost two months; weight 25½ pounds (11.5 Kg.)	None	In institution eight months; no signs of rickets; died of pneumonia and mastoid; liver found markedly congested
46–46				None	
50–50				None	
50–54	1.0			None	
50–40				None	
44–42					
60–80	2.0	5 months	Breast fed for 3 months	Moderate	Birth weight 7½ pounds (3.4 Kg.); Died of pneumonia
50–60				None	
40–40				Slight	
90–100	2.0	2½ months	Breast fed for 3 weeks; cod liver oil few days; ultraviolet irradiation nine times	Slight or moderate	Premature birth weight 3 pounds, 13 ounces (1.7 Kg.)
40–110				Moderate (?)	
60–80				Moderate	
40–40	1.0	19 days		None	Liver extracted with ether; 0.6 cc. = 1 Gm.
50–52				None	
40–30	0.5	Adult 27 years old	General diet	None	
40–40				None	
60–50				None	
56–54	1.0			None	
60–55				None	
50–40				Slight	
40–50	2.0			Moderate	
36–50				Very Slight	
30–52	1.0	Adult	General diet	None	
30–54				None	
30–40				None	
30–50	1.0	Adult	General diet	None	
28–46				None	
30–41				None	

A comparison of the two sets of tests renders it evident that there was no essential difference in potency between the livers of the full-term and of the premature infants. The fact that the liver of a premature infant, after only four and one-half months' gestation and but 27 cm. in length, showed protective properties indicates that the theory of a prenatal deficiency of antirachitic "vitamin" does not have any foundation.

Table 3 contains data concerning the livers of infants who lived for a few weeks or months after birth, as well as data in regard to a few

adults. These may be summarized by the statement that after a short period of extra-uterine life, a somewhat greater amount of antirachitic factor was found than at the time of birth. The difference, however, was not great or constant. Although this aspect is foreign to the question that we set ourselves, it may be of interest to call attention to some details in connection with these cases. The first infant noted in the table, aged 5 months, was an exceptionally fine infant, and weighed 19 pounds (8.6 Kg.) at the time of death. The infant had been nursed for three months. It died of an acute illness that lasted only a few days—pneumonia complicated by meningitis. The biologic tests of the liver showed, in this instance, the greatest amount of antirachitic factor. Possibly this was due to the mother's milk; the infant had not received cod liver oil or been subjected to a great intensity of sunlight. The second infant on the list was also a splendid child, and weighed 25½ pounds (11,500 Gm.) when 11½ months old, at the time of its death, which likewise was due to pneumonia. The liver in this case was found lacking in antirachitic factor. It should be added that this child had received three teaspoonfuls of cod liver oil daily for almost two months previous to the time of death. Evidently, there are individual differences in the storage of the specific factor in the liver. Still another case may be mentioned—that of an infant, aged 2½ months. This infant was nursed for three weeks, had received extremely small amounts of cod liver oil for a few days and nine irradiations from the mercury-vapor lamp. The liver assayed rather high, but was tested only at the upper level; 2 Gm. was fed daily. The case is mentioned because the infant was born prematurely, and probably acquired its antirachitic store during its brief postnatal life.[2]

A few assays of the livers of adults are appended. They are of interest from a general nutritional and dietary point of view and not in relation to rickets. We did not expect to find a large amount of the antirachitic factor in these organs, as the diet of adults is exceedingly poor in this nutritional factor, with the exception of yolk of egg. It will be noted that only one of three livers assayed, gave evidence of antirachitic potency when 1 Gm. was fed daily, an amount which sufficed to induce calcification of the epiphyses in the tests made on premature infants. It would seem that the liver of man, as of cattle and of pigs, is practically devoid of this specific factor.

---

2. In view of the fact that it is commonly believed that rickets plays a prominent rôle in the susceptibility, incidence and mortality of pneumonia, it would be of interest to make biologic assays of the livers of infants who died of this disease and who had received adequate amounts of cod liver oil, irradiated ergosterol or of direct irradiation during their lifetime. Would these organs be found devoid of the antirachitic factor?

SUMMARY

It has been taken for granted that variation in predisposition to rickets is due largely to variability in the store of the antirachitic factor which infants bring with them into the world, the store being magazined in the liver. In order to subject this hypothesis to the test of experiment, we carried out a biologic assay of the livers of a series of infants. As it is well established that premature infants have the greatest predisposition to rickets—in fact, few of them are spared even when nursed and given cod liver oil early in life—one would expect a definite lack of the antirachitic factor in this group as compared with infants born at full term. A comparison of this kind should serve to bring into sharp relief the assumed prenatal "vitamin" deficiency of the susceptible infant.

Our study showed, on the contrary, that the livers of premature infants contain as much of the antirachitic factor as those of infants born at term. In view of this result, it would seem that, in general, individual predisposition to this disorder does not depend on prenatal hoarding of this "vitamin," but on other factors. Differences in the percentage of ash in the skeleton at birth and in the rate of growth in the early months of postnatal life undoubtedly are of importance, but in all probability deeper biologic influences, of which we have no knowledge, also play a rôle.

STIMULATION OF GROWTH OF SCHOOL CHILDREN BY SMALL SUPPLEMENTARY FEEDINGS*

AGNES FAY MORGAN, PH.D.
AND
LYDIA WARREN, B.S.
BERKELEY, CALIF.

A number of studies have been made by the Household Science Department of the University of California on the effect of supplementary lunches on the growth of school children. The first of these by Chaney [1] was carried on in 1923 in the Claremont school in Oakland, Calif., with children of families belonging for the most part to the higher economic and social groupings. This study covered the growth and physical status of 109 and 140 children observed during two eight-week periods of supplementary feeding. One orange and two graham crackers or half a pint of whole milk and two graham crackers were used, and a definite advantage was seen in the groups fed on oranges. All children in these groups were 7 per cent or more underweight for height and age according to the Wood scale of 1920. All groups of children fed with oranges, orange and milk, orange-ade or milk made much larger gains than did those in the control group who did not have a supplementary feeding.

In 1925, a second study [2] was made in the hope of discovering more definite information concerning the value of these extra feedings by comparing groups of children known to have the same basal diet. Accordingly, forty-seven children living in the California State School for the Deaf and Blind at Berkeley were divided into four groups. The children in three of the groups were given supplementary feedings daily, consisting, respectively, of an orange, half a pint of whole milk and four pulled figs. The children in the fourth group, the controls, were not given supplementary feedings. Physical measurements were made once a month during the test period of from fourteen to eighteen weeks. In most cases, these measurements included basal metabolism, plasma alkaline reserve, vital capacity and circumference and expansion of chest, as well as the usual weight and height measurements. The

* Received for publication, June 28, 1928.

* From the Laboratory of Household Science, the University of California.

1. Chaney, M. S.: A Comparison of the Value of Milk and Oranges as Supplementary Lunch for Underweight Children, Am. J. Dis. Child. **26**:337 (Oct.) 1923.

2. Morgan, A. F.; Hatfield, G. D., and Tanner, M. A.: A Comparison of the Effects of Supplementary Feeding of Fruits and Milk on the Growth of Children, Am. J. Dis. Child. **32**:839 (Dec.) 1926.

growth shown by the three groups given extra feedings was much alike, and was in excess of that found in the control group. The children fed on oranges and on milk made similar gains in most measurements, but the children fed on figs showed unusual increases in all measurements of the chest. Since the house diet included a quart of milk daily per child, any unusual gain due to the supplementary feeding of milk was hardly to be expected.

The following year, a short study [3] of the feeding of similar amounts of figs and oranges to thirty-six high school students was made at Livermore, Calif. An attempt was not made to influence the home diet of these subjects, from 14 to 18 years of age, all but eight of whom were 7 per cent or more underweight for height, according to the Baldwin-Wood standard. The groups fed on oranges and on figs made practically equal gains in weight during the eight weeks period of feeding, and more than twice as much as the control group.

It seemed plain that a definite superiority as supplementary lunch could not be assumed for either milk or the fruits, and that differences in the nature of the home diet of the children might well account for whatever differences were observed in all these tests. It seemed desirable therefore to make an examination of the effect of certain foods on the growth of children of a lower economic status than those previously studied. The Prescott School in Oakland, Calif., was selected for this purpose, since the majority of children attending there came from homes of low level incomes or from a nearby orphanage. About one fifth of the children were negroes, and the others were of Italian, Portuguese and mixed American stock. The proportion of undernutrition in this school is high, and supplementary feeding of milk and orange juice has been in effect for some time as a school measure.

The children selected for feeding were chosen from among those sent to us by the teachers as obviously underweight or otherwise in a poor state of nutrition. About 300 children were completely examined, and some 200 of these were chosen for the study. Weight, standing height, sitting height, circumference and expansion of the chest and the vital capacity were observed with the usual precautions for accuracy as described by Baldwin.[4] All measurements were made by two observers and checked at once or the next day by two other observers. The difficulty of obtaining accurately reproducible figures for sitting height was particularly obvious, as was the futility of attempting to make measurements of the chest over clothing. Only the weight and standing

3. Morgan, A. F., and Tanner, M. A.: Supplementary Feeding of School Children, Am. J. Dis. Child. **33**:404 (March) 1927.

4. Baldwin, B. T.: The Use and Abuse of Weight-Height-Age Tables as Indexes of Health and Nutrition, J. A. M. A. **82**:1 (Jan. 5) 1924.

height figures are therefore used in this comparison. Only 177 complete records were obtained.

An attempt was not made to influence the general diet of the children. Four groups of children with similar nutritive condition, with the exception of the control group which was made up of comparable children, were formed from among the children selected. To each child in one of the groups was given one large orange [5] daily, to a second group half a pint of milk, to a third group two soda crackers with a sugar filling and to the fourth group nothing. The cracker sandwich was made with a filling of powdered sugar, corn oil, dextrose and a little cocoa flavoring, and was used in the hope of discovering the effect of the addition of food calories without notable protein, vitamin or mineral content. Something of the same sort was tried a few years ago by McCollum [6] at an institution for negro children with inconclusive results.

The supplementary feedings were given every school day from April 4 to June 10, 1927, with the exception of one week of vacation

TABLE 1.—*Supplementary Lunches used in Ten Weeks' Test*

Group	Number of Children in Group	Supplementary Lunch	
		Food	Caloric Value
Milk	50	One-half pint pasteurized whole milk	160
Orange	40	One large orange	80
Cracker	41	Two soda crackers with sugar filling	125
Control	46	Nothing	...

in April. The total period of feeding was ten weeks. The children were given the usual physical examination with special attention to nose, throat and chest, and only those who were normal were retained on the list of children who were given supplementary feedings. All examinations were repeated at the end of the feeding period with proper attention to exact repetition of measuring conditions. In table 1 the caloric value of the supplementary lunches is shown, and in table 2 the condition of nutrition of the children at the beginning of the experiment. It will be noted that the average state of nutrition of the children in the three groups which received the foods was much alike, and that approximately half of each group was more than 7 per cent underweight for height and age judged by the Baldwin-Wood standard. The control group which did not receive supplementary feeding contained only three children, or 6 per cent of the group,

5. The oranges were supplied by the California Fruit Growers' Exchange.

6. McCollum, E. V.; Parsons, H. T., and Kalmbach, E.: The Nutritional Value of Milk, Government Printing Office, 1924; quoted by McCollum, E. V., and Simmonds, N.: The Newer Knowledge of Nutrition, ed. 3, New York, The Macmillan Company, 1925, p. 541.

who were more than 7 per cent underweight by the same standard. This difference in composition of the groups was effected in order to provide the more favorable conditions for the controls and therefore to allow more confidence in the validity of any increase in rate of growth which might occur during the supplementary feeding. Particular importance is not attached to the amount of underweight shown by these children, the object of the study being a comparison of growths effected rather than the attainment of any particular standard by the subjects.

TABLE 2.—*Condition of Nutrition of Children Observed*

Group	Number			Age			0 to 7 Per Cent Underweight		7.1 to 12 Per Cent Underweight		More Than 12.1 Per Cent Underweight	
	Boys	Girls	Total	6-9 Years	10-11 Years	12-14 Years	Number	Percentage of Group	Number	Percentage of Group	Number	Percentage of Group
Milk	29	21	50	12	19	19	21	42.0	23	46.0	6	12.0
Orange	26	14	40	20	15	5	20	50.0	17	42.0	3	7.5
Cracker ...	16	25	41	9	20	12	21	51.2	14	34.1	6	14.6
Control ...	27	19	46	24	14	8	43	93.4	2	4.3	1	2.2

TABLE 3.—*Growth of Children Given Supplementary Lunches*

Age Group Years	Supplementary Lunch	Number of Children	Average Percentage Underweight at Beginning	Average Gain in Weight in Ounces	Average Percentage of Expected Gain in Weight	Average Increase in Height in ⅛ Inches	Percentage Making Gains in Height
6-9	Milk	12	5.3	24	155	3.4	75
	Orange	20	6.4	14	86	4.3	85
	Crackers	9	4.8	18	137	3.9	67
	Control	24	0.4	11	77	3.3	70
10-11	Milk	19	7.2	27	139	3.8	79
	Orange	15	7.4	14	73	4.6	93
	Crackers	20	8.4	19	88	3.6	75
	Control	14	+0.7	— 4	— 27	3.4	71
12-14	Milk	19	7.4	20	61	3.4	73
	Orange	5	8.4	15	57	4.4	80
	Crackers	12	7.6	36	107	3.0	67
	Control	8	+0.9	— 3	— 11	2.9	62
All children	Milk	50	6.5	24	104	3.6	76
	Orange	40	7.6	14	76	4.4	87
	Crackers	41	7.0	24	102	3.5	70
	Control	46	0.1	4	22	3.3	69

The amount of growth in weight and standing height is shown in table 3. As may be seen, the three groups that were given supplementary feedings in all cases made considerably greater gains in weight than did the control group. The children fed on milk and the children fed on crackers apparently gained in somewhat the same proportion, the children fed on oranges gained somewhat less rapidly. The gains in height are not strikingly different in any of the groups, but it may be significant that 87 per cent of all the group fed on oranges made gains in height and also made the largest average amount of gain, 76 per cent of the group fed on milk gained, and 70 and

69 per cent of the group fed on crackers and the control groups. These children seemed to profit by additional calories as to weight, but to obtain in addition stimulation to height or skeletal growth from the added orange juice and milk.

A striking advantage in amount of gain in weight is shown by the girls over the boys of all groups as indicated in table 4. This is particularly evident in the group fed on oranges of which an active baseball-playing set of boys formed a large part. A similar but less marked advantage in increased height is seen in the girls of the three groups that were given supplementary feedings.

TABLE 4.—*Growth of Boys and Girls as Influenced by Supplementary Feeding*

Group	Age Years	Number		Average Gain in Weight in Ounces		Average Percentage of Expected Gain Attained		Average Increase in Height in 1/8 Inches		Percentage Making Gains in Height	
		Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
Milk	6- 9	5	7	18.4	29.0	115	185	3.4	3.4	80	71
	10-11	13	6	25.2	32.7	149	142	3.6	4.5	77	83
	12-14	11	8	18.5	21.5	60	64	3.4	3.5	64	87
All ages		29	21	21.5	27.2	97	110	3.4	3.7	72	81
Oranges	6- 9	13	7	13.4	17.1	76	107	4.5	4.0	93	71
	10-11	8	7	4.7	25.0	31	103	3.3	6.3	87	100
	12-14	5	0	15.6	....	57	...	4.4	...	80	...
All ages		26	14	7.3	21.0	59	108	4.1	5.1	89	86
Crackers	6- 9	4	5	25.0	13.6	196	96	2.5	5.0	50	80
	10-11	7	13	9.7	25.0	58	99	2.9	4.0	57	85
	12-14	5	7	18.5	48.6	60	136	1.2	4.4	40	86
All ages		16	25	16.2	29.4	83	115	2.2	4.3	50	84
Controls	6- 9	10	14	17.3	6.9	106	52	3.3	3.4	70	71
	10-11	9	5	— 8.4	2.4	— 61	11	3.9	2.8	78	60
	12-14	8	0	— 3.0	....	— 11	...	2.9	...	62	..
All ages		27	19	2.7	5.6	14	30	3.3	3.2	70	68

Large deviations from the arithmetical means of growth summarized in the tables were found. It was not to be expected with the small number of children studied that statistical treatment of the observations would be valid. There were even stationary or declining weights for instance in all groups. It is interesting that seven such cases should occur in each of the three groups that were given supplementary lunches and twenty-four in the control group. The total loss in all measurements is subtracted from the total increase in each group, and the net gain thus obtained is averaged.

It seems clear that the children observed, whether from lack of opportunity or appetite, were underfed as to total calories, and that the supplementary lunches which provided the most extra calories were most effective in meeting this condition. Obviously, any addition to the diet at school is an advantage and is not counterbalanced by corresponding decrease in intake of food at home. As shown in table 5,

this result is in contrast with that found by Chaney [1] at the Claremont School and by Morgan, Hatfield and Tanner [2] at the California State Schools for the Deaf and Blind. In the former study, the children who received supplementary feedings made far larger gains than the controls, and the group fed on oranges made noticeably more gains in weight than the group fed on milk. In the latter study, the gain in weight by the controls was not much different from the groups given extra feedings, although gains in other measurements were superior in the latter. In the study at the Livermore High School,[3] again the groups given supplementary feedings far outstripped the controls, but here only fruits were used, so that nothing was learned about the

TABLE 5.—*Comparison of Weights of School Children in Four Schools as Affected by Supplementary Lunches*

School	Supplementary Lunch	Number of Children	Ages Years	Length of Period Weeks	Average Gain in Kilograms Per Week	Average Percentage of Expected Gain Attained	Economic Status
Claremont School (winter)	Milk	28	6	8	0.118	206	Excellent
	Orange	10	to	..	0.174	241	
	Control	34	14	..	0.085	128	
Claremont School (spring)	Milk	37	6	8	0.114	179	Excellent
	Orange	38	to	..	0.144	218	
	Control	35	14	..	0.074	108	
California State School for Deaf and Blind	Milk	13	6	14	0.110	177	State institution, diet of high quality
	Orange	13	to	..	0.087	177	
	Fig	10	14	..	0.074	167	
	Control	11	..	..	0.097	169	
Livermore High School	Orange	17	14	8	0.093	290	Fair
	Fig	8	to	..	0.092	287	
	Control	11	18	..	0.043	102	
Prescott School	Milk	50	6	10	0.068	104	Poor
	Orange	40	to	..	0.041	76	
	Crackers	41	14	..	0.040	102	
	Control	46	..	..	0.011	22	

comparative value of fruits and milk. In the present study at the Prescott School, a more striking difference is shown between groups given supplementary lunches and control groups than in any of the others. Milk and cracker-sugar sandwiches appear to be equally helpful in promoting increases in weight, and oranges less valuable possibly in proportion to the lower number of calories which they furnished. This contrast is probably the result of an equally striking contrast in home diet as influenced by economic condition and amount of parental care received by the children studied in the various schools. Clearly, supplementary lunches at school are worth while for all undernourished children, but the best type of food used for this purpose cannot be chosen without careful consideration of the prevailing home conditions. A preliminary study such as is here reported, of from eight to twelve weeks, involving the use of three or four kinds of foods, should precede

such choice in any school. In the Prescott School, for instance, we recommended that only milk or crackers be served and that if possible all children fed be given both.

SUMMARY

1. One hundred and seventy-seven children in attendance at a public school in Oakland, Calif., were chosen for measurement and supplementary feeding. The economic status of the families from which these children came is relatively low. About one half of the children were 7 per cent or more underweight for height.

2. Four groups were formed, one of which did not receive extra food and the others of which received half a pint of milk, one orange and a small cracker sandwich, respectively, each school day for ten weeks. Changes in weight and standing height were observed at the end of the period.

3. The children in all three groups given supplementary feedings made large gains in growth as compared with the controls. The groups fed on milk and crackers gained weight at about the same rate, and the group fed on oranges somewhat less. The group fed on oranges, however, made better gains in height than the others.

4. The girls in all groups and at all ages gained more in both weight and height than the boys.

5. The mere addition of extra calories furnished a measurable advantage for these children.

6. These results are compared with those found in former studies, and the inference is drawn that choice of supplementary lunch for a given group of children should be made only after careful consideration of home conditions and preferably after such preliminary observation as is here described.

METABOLISM OF UNDERNOURISHED CHILDREN

VI. CALORIC BALANCE *

CHI CHE WANG, PH.D.

MARGARET FRANK, B.S.

AND

MILDRED KAUCHER, B.S.

CHICAGO

In the previous papers [1] of this series, various phases of the metabolism of undernourished children have been discussed. In this paper, data concerning the caloric balance of ten normal and twenty-six undernourished children are given. In all, fifty-two sets of tests were made, some of the children appearing two, three or four times, usually since they remained in the hospital and were gaining in weight. Most of these children have also been used as subjects in our previous investigations.

Practically nothing can be found in the literature on the subject of the metabolism of the undernourished child of school age. Utheim [2] and Fleming [3] have published data on the metabolism of athreptic infants, Utheim drawing the conclusion that in severe cases the infant's capacity for the absorption of food material was markedly decreased. This improved rapidly, however, as the infant gained in weight. Fleming, on the other hand, believes that his own data, and also those of Utheim, if reconsidered, show that the absorbing power of the athreptic infant is fully equal to, or greater than, that of the normal child. His thesis is that the caloric output is proportional to the weight of the feces and

---

* Received for publication, July 16, 1928.

* From the Nelson Morris Institute for Medical Research and the Sarah Morris Hospital for Children of the Michael Reese Hospital. Aided by a fund contributed jointly by the Elizabeth McCormick Fund, Mrs. Francis Neilson, Mrs. Gusta Morris Rothschild and Mr. Melville M. Rothschild.

1. Wang, C. C.; Frank, M.; Hays, B. B., and Kern, R.: Metabolism of Undernourished Children: I. Gain in Weight, Am. J. Dis. Child. **32**:63 (July) 1926; Metabolism of Undernourished Children: II. Basal Metabolism, Am. J. Dis. Child. **32**:350 (Sept.) 1926; Metabolism of Undernourished Children: III. Urinary Nitrogen with Special Reference to Creatinine, Am. J. Dis. Child **32**:360 (Sept.) 1926. Wang, C. C.; Kaucher, M., and Frank, M.: Metabolism of Undernourished Children: IV. Calcium Metabolism, in press. Wang, C. C.; Hawks, J. E., and Hays, B. B.: Metabolism of Undernourished Children: V. Protein Metabolism, Am. J. Dis. Child. **35**:968 (June) 1928.

2. Utheim, K.: Metabolism Studies in Infants Suffering from Chronic Nutritional Disturbances (Athrepsia), Am. J. Dis. Child. **22**:329 (Oct.) 1921.

3. Fleming, G. B., and Hutchison, H. S.: A Study of the Metabolism in the Undernourished Infant, Quart. J. Med. **17**:339 (July) 1923.

independent of the intake, the mean weight of the feces being approximately the same for both normal and athreptic infants; the percentage absorption, therefore, is inversely proportional to the intake. He therefore believes that had the caloric intake been the same in the two groups of infants studied by Utheim, the actual and the percentage absorption would have been the same.

EXPERIMENTAL WORK

The same technic of feeding and of the collection of food and excreta was used in this series of experiments as has already been reported in previous papers.[4] To determine the caloric intake the children were fed a weighed diet, as usual, and the energy value of the diet was computed according to values published by Rose [5] in her "Laboratory Manual for Dietetics." The caloric value of the excreta was determined in the adiabetic bomb calorimeter designed by Benedict.[6] The feces were dried and ground as described in previous papers, and pellets were made from the powder for use in the calorimeter. The urine was prepared according to the method of Higgins and Benedict.[7] Determinations were made in triplicate and the average taken of the two most closely agreeing results. In the case of urine, samples were collected and the caloric value determined daily. The figures given in the tables represent the average of a three day period. The feces were collected over a three day period, and one triplicate determination made for the three days.

As in our previous work, the children were divided into five groups as follows: group 1, vigorous normal children; group 2, normal in weight only; group 3, from 5.1 to 10 per cent underweight; group 4, from 10.1 to 15 per cent underweight, and group 5, more than 15 per cent underweight.

COMMENT

An examination of tables 1 to 5 will show that the average total caloric intake, and also the average total caloric loss, was higher in the group of normal children than in the groups in which they were underweight, but that when computed on the basis of per kilogram of body weight both the average intake and the average output were lower in this group. The total calories utilized were higher for the normal than for the groups of undernourished children, but the percentage of the total caloric intake utilized was practically the same in all groups, varying from 86.9 to 93.8 per cent, with an average of 91.6 per cent.[8] The calories utilized

4. Wang; Frank; Hays, and Kern (footnote 1, first reference).

5. Rose, M. S.: A Laboratory Handbook for Dietetics, revised ed., New York, The Macmillan Company, 1925.

6. Benedict, F. G., and Higgins, H. L.: An Adiabatic Calorimeter for Use with the Calorimetric Bomb, J. Am. Chem. Soc. **32**:461 (April) 1910.

7. Higgins, H. L., and Benedict, F. G.: Some Energy Factors of the Urine Excreted After Muscular Exercise, Am. J. Physiol. **28**:291 (Sept.) 1911.

8. These figures are in close agreement with those quoted by Holt for healthy children. Holt, L. E., and Fales, H. L.: Food Requirements for Children: II. Protein Requirements, Am. J. Child. Dis. **22**:371 (Oct.) 1921.

TABLE 1.—*Caloric Balance of Vigorous Normal Children*

			Weight		Intake	Output						Utilization			Heat Production per 24 Hrs.					
						Urine		Feces		Total						Excess Over Basal				
Name	Sex	Age	Kg.	Variation from Normal	24 Hrs.	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	Percentage	Basal Calories	Total Calories	Per Kg. Calories	Urinary Nitrogen Gm.	Calory Nitrogen Ratio	Gain in Weight per 24 Hrs. Gm.
M. S.	M	9	33.5	+17.1	2,578	82	2.4	94	2.8	175	5.2	2,403	71.7	93.2	1,208	1,195	35.7	10.1	8.12	28
M. F.	F	8	27.7	+ 8.6	2.061	82	3.0	90	3.2	172	6.2	1,889	68.2	91.7	1,030	859	31.0	9.4	8.72	31
L. F.	F	10	26.9	+ 3.9	1,929	81	3.1	92	3.4	174	6.5	1,755	62.5	91.0	1,054	701	26.1	10.5	7.71	00
M. H.	F	9	23.9	+ 3.2	1,816	72	3.0	80	3.4	152	6.4	1,664	69.6	91.6	1,049	615	25.7	10.9	6.61	00
S. A.	F	10	29.2	+ 1.4	2.260	84	2.9	88	3.0	171	5.9	2,089	71.5	92.4	1,092	997	34.1	11.3	7.43	67
L. L.	M	10	34.0	+ 1.2	2,617	84	2.5	78	2.3	163	4.8	2,454	72.2	93.8	1,267	1,187	34.9	10.7	7.81	11
B. L.	F	12	33.0	+ 1.2	2,293	94	2.9	119	3.6	213	6.5	2,086	63.2	91.0	1,067	1,019	30.9	12.4	7.58	25
F. J.	F	11	28.2	+ 0.6	2,016	83	2.9	104	3.7	186	6.6	1,830	64.9	90.8	1,127	703	24.9	10.6	7.83	13
R. G.	F	10	28.6	— 3.4	2,016	79	2.7	122	4.3	201	7.0	1,815	63.5	90.0	960	835	29.2	10.5	7.52	00
H. S.	F	9	27.2	— 3.5	2,030	61	2.2	106	3.9	166	6.1	1,864	68.5	91.8	996	868	31.9	10.7	5.70	11
Average		9.8	29.2	+ 3.0	2.102	80	2.8	97	3.4	177	6.1	1,922	65.4	91.7	1,087	835	28.2	10.7	7.51	18.6

TABLE 2.—*Caloric Balance of Children Normal in Weight Only*

			Weight		Intake	Output						Utilization			Heat Production per 24 Hrs.					
						Urine		Feces		Total						Excess Over Basal				
Name	Sex	Age	Kg.	Variation from Normal	24 Hrs.	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	Percentage	Basal Calories	Total Calories	Per Kg. Calories	Urinary Nitrogen Gm.	Calory Nitrogen Ratio	Gain in Weight per 24 Hrs. Gm.
E. F.	F	6	19.3	+ 4.9	1,713	58	3.0	84	4.4	142	7.4	1,571	81.4	91.7	927	644	33.4	8.0	7.25	24
H. D.	M	5	13.9	± 0.0	1,267	52	3.7	52	3.7	104	7.4	1,159	83.4	91.5	802	357	25.7	5.9	8.81	40
E. G.	F	6	18.1	— 1.1	1,628	52	2.9	95	5.3	147	8.2	1,481	81.8	91.0	976	505	27.9	6.8	7.65	17
N. G.	M	7	16.9	— 1.2	1,556	46	2.7	63	3.7	109	6.4	1,447	85.6	93.0	1,052	395	23.4	7.5	6.13	25
E. G.	F	6	17.5	— 1.7	1,653	53	3.0	52	3.0	105	6.0	1,548	98.5	93.7	976	572	32.7	6.3	8.41	60
E. G.	F	6	17.5	— 1.7	1,620	41	2.5	96	5.5	140	8.0	1,480	84.6	91.4	976	502	28.7	5.9	6.95	00
G. L.	F	13	34.2	— 3.3	2,171	51	1.5	106	3.1	157	4.6	2,010	58.9	92.8	1,182	828	24.3	7.1	7.18	57
N. G.	M	7	16.5	— 3.5	1.553	50	3.0	94	5.7	144	8.7	1,409	85.4	90.7	1,052	357	21.6	6.5	7.69	64
I. Ba.	M	10	30.5	— 4.1	1,722	55	1.8	111	3.6	166	5.4	1,556	51.0	91.4	1,143	413	13.5	7.3	7.53	00
E.F.	F	6	17.3	— 4.9	1,798	62	3.6	80	4.6	142	8.2	1,650	95.7	92.1	927	729	42.1	7.7	8.05	44
Average		7.2	20.2	— 1.6	1,668	52	2.8	83	4.3	136	7.0	1,532	79.6	91.9	1,001	530	27.3	6.9	7.57	33.1

TABLE 3.—*Caloric Balance of Children from 5.1 to 10 per cent Underweight*

Name	Sex	Age	Weight		Intake		Output						Utilization			Heat Production per 24 Hrs.					
							Urine		Feces		Total						Excess Over Basal		Urinary Nitrogen Gm.	Calory Nitrogen Ratio	Gain in Weight per 24 Hrs. Gm.
			Kg.	Variation from Normal	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	Percentage	Basal Calories	Total Calories	Per Kg. Calories			
H. F.	M	9	26.7	— 5.3	2,026	75.9	61	2.3	106	4.0	167	6.3	1,859	69.6	91.7	996	863	32.3	....	.....	...
E. B.	F	5	15.8	— 5.4	1,674	106.0	57	3.6	124	7.9	181	11.5	1,493	94.5	89.2	853	640	40.5	5.4	10.56	146
J. G.	M	8	22.4	— 5.9	1,946	86.9	63	2.8	96	4.3	156	7.1	1,787	79.9	91.9	1,117	670	29.9	7.4	8.51	24
L. C.	F	6	18.7	— 7.9	1,691	90.4	48	2.6	127	6.8	174	9.4	1,517	81.1	93.4	868	649	34.7	6.4	7.50	14
R. G.	M	6	16.3	— 7.9	1,707	104.7	48	3.0	70	4.3	118	7.3	1,589	97.5	93.1	966	623	38.2	6.3	7.62	21
J. U.	F	9	25.9	— 8.2	2,042	78.8	66	2.5	68	2.6	134	5.1	1,908	73.7	93.4	1,023	885	34.2	6.2	10.64	...
E. G.	F	6	16.3	— 8.4	1,147	70.4	44	2.7	54	3.3	98	6.0	1,049	64.4	91.5	984	65	4.0	6.4	6.88	00
B. G.	F	9	20.5	— 8.5	1,705	83.2	72	3.5	100	4.9	172	8.4	1,533	74.8	87.9	938	595	29.0	7.7	9.35	00
L. C.	F	6	18.4	— 9.4	1,721	93.5	51	2.8	103	5.6	154	8.4	1,567	85.2	91.1	868	699	38.0	6.7	7.61	17
T. F.	M	10	23.4	— 9.8	1,854	79.2	79	3.4	93	4.0	172	7.4	1,682	71.9	90.7	1,118	564	24.1	9.5	8.32	36
S. K.	M	10	27.5	— 9.8	2,307	83.9	81	3.0	116	4.2	198	7.2	2,109	76.7	91.4	1,055	1,054	38.3	11.4	7.11	33
Average		8.6	21.1	— 7.9	1,711	86.6	61	2.9	96	4.7	157	7.7	1,645	79.0	91.6	961	664	31.2	7.3	8.41	29.1

TABLE 4.—*Caloric Balance of Children from 10.1 to 15 per cent Underweight*

			Weight		Intake	Output						Utilization			Heat Production per 24 Hrs.					
						Urine		Feces		Total						Excess Over Basal				
Name	Sex	Age	Kg.	Variation from Normal	24 Hrs.	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	Percentage	Basal Calories	Total Calories	Per Kg. Calories	Urinary Nitrogen Gm.	Calory Nitrogen Ratio	Gain in Weight per 24 Hrs. Gm.
H. L.	F	8	19.3	—10.6	1,514	83	4.3	116	6.0	199	10.3	1,315	68.1	86.9	1,047	268	13.9	6.8	12.21	34
I. Bo.	M	9	20.7	—10.7	1,996	55	2.7	102	4.9	157	7.6	1,839	88.8	92.1	1,030	803	38.8	5.8	9.48	..
H. G.	M	11	27.5	—10.7	1,979	59	2.1	96	3.5	155	5.6	1,824	66.3	92.2	1,083	741	26.9	8.3	7.11	65
L. C.	F	6	18.0	—11.3	1,604	46	2.5	87	4.9	133	7.4	1,471	81.7	91.7	904	567	31.5	6.6	6.97	50
J. M.	M	13	27.5	—12.1	2,076	76	2.8	84	3.1	160	5.9	1,916	69.7	92.3	1,248	668	24.3	9.4	8.09	58
S. K.	M	10	26.8	—12.1	2,249	73	2.7	118	4.4	191	7.1	2,158	80.5	91.9	1,086	1,072	46.0	12.3	5.94	68
N. G.	M	7	15.0	—12.3	1,673	54	3.6	81	5.4	135	9.0	1,538	102.5	91.9	1,052	486	32.4	7.0	7.71	43
R. D.	F	10	19.1	—12.3	1,819	51	2.7	75	3.9	126	6.6	1,693	88.7	93.1	1,021	672	35.2	6.0	8.50	..
E. F.	F	6	15.4	—13.5	1,243	52	3.4	64	4.2	116	7.5	1,127	73.2	90.7	868	260	16.9	6.6	7.88	33
B. G.	F	8	19.1	—13.6	2,081	66	3.5	88	4.6	154	8.1	1,927	100.8	92.6	980	947	49.6	5.8	11.38	29
R. B.	M	6	16.4	—14.1	1,587	61	3.7	100	6.1	160	9.8	1,427	87.6	89.9	903	434	26.5	6.5	9.38	16
Average		8.6	20.4	—12.1	1,630	61	3.1	92	4.7	153	7.7	1,658	82.5	91.4	1,029	629	30.6	7.4	8.60	44

TABLE 5.—*Caloric Balance of Children More than 15 per cent Underweight*

			Weight		Intake	Output						Utilization			Heat Production per 24 Hrs.					
						Urine		Feces		Total						Excess Over Basal				
Name	Sex	Age	Kg.	Variation from Normal	24 Hrs.	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	24 Hrs.	Per Kg. of Body Weight	Percentage	Basal Calories	Total Calories	Per Kg. Calories	Urinary Nitrogen Gm.	Calory Nitrogen Ratio	Gain in Weight per 24 Hrs. Gm.
L. C.	F	6	17.1	—15.8	1,309	40	2.3	78	4.6	118	6.9	1,191	69.7	91.0	904	287	16.8	5.0	8.00	00
D. Y.	F	7	17.3	—16.0	1,540	53	3.1	85	4.9	138	8.0	1,402	82.1	92.2	956	446	25.8	7.0	7.57	30
E. F.	F	6	15.2	—16.5	1,680	52	3.4	99	6.5	151	9.9	1,529	100.6	91.0	661	868	57.1	6.1	8.52	20
S. K.	M	10	25.4	—16.7	2,312	75	3.0	143	5.6	218	8.6	2,094	82.4	90.5	1,086	1,008	39.7	11.9	6.30	10
A. S.	F	10	17.8	—16.8	1,608	60	3.4	67	3.8	127	7.2	1,481	83.2	92.2	952	529	29.7	8.2	7.32	32
I. Be	F	11	26.0	—17.2	1,838	51	2.0	109	4.2	160	6.2	1,678	64.5	91.3	1,098	580	22.3	5.8	8.79	..
A. S.	F	10	17.4	—18.7	1,625	57	3.3	53	3.0	110	6.3	1,515	87.1	93.2	952	563	32.4	7.0	8.14	18
A. S.	F	10	16.9	—19.2	1,675	61	3.6	71	4.2	131	7.8	1,544	91.4	92.1	952	612	36.2	7.3	8.36	40
L. P.	F	8	22.6	—19.8	1,633	57	2.5	89	4.0	146	6.5	1,487	65.8	91.1	1,004	483	21.4	6.6	8.64	21
A. G.	F	8	17.9	—25.7	1,189	55	3.1	85	4.7	140	7.8	1,049	58.6	88.2	784	265	14.8	6.6	8.33	16
Average		8.6	19.4	—18.2	1,640	56	3.0	88	4.6	144	7.5	1,497	78.5	91.3	935	564	29.6	7.2	8.00	20.8

above the basal requirement were higher per twenty-four hours in the normal children than in the groups of undernourished children, but approximately the same per kilogram of body weight in all groups. With the exception of four children, having values of 10.6, 10.6, 12.2 and 11.4, respectively, the ratios of calories to nitrogen are all within the normal

TABLE 6.—*Caloric Balance per Twenty-Four Hours*

Name	Sex	Age	Weight		Intake	Output			Utilization	Percentage Utilization
			Kg.	Deviation from Normal		Urine	Feces	Total		
L. L.	M	10	34.0	+ 1.2	2,617	84	78	163	2,454	91.0
M. S.	M	9	33.5	+17.1	2,578	82	94	175	2,403	93.2
S. K.	M	10	26.8	—12.1	2,349	73	118	191	2,158	91.9
S. K.	M	10	25.4	—16.7	2,312	75	143	218	2,094	90.5
S. K	M	10	27.5	— 9.8	2,307	81	116	198	2,129	91.4
B. L.	F	12	33.0	+ 1.2	2,298	94	119	213	2,086	91.0
S. A.	F	10	29.2	+ 1.4	2,260	84	88	171	2,089	92.4
G. L.	F	13	34.2	— 3.3	2,171	51	106	157	2,016	92.8
B. G.	F	8	19.1	—13.6	2,081	66	88	154	1,927	92.6
J. M.	M	13	27.5	—12.1	2,076	76	84	160	1,916	92.3
M. F.	F	8	27.7	+ 8.6	2,061	82	90	172	1,889	91.7
J. U.	F	9	25.9	— 8.2	2,042	66	68	134	1,908	93.4
H. S.	F	9	27.2	— 3.5	2,030	61	106	166	1,864	91.8
H. F.	M	9	26.7	— 5.3	2,026	61	106	167	1,859	91.7
F. J.	F	11	28.2	+ 0.6	2,016	83	104	186	1,830	90.8
R. G.	F	10	28.6	— 3.4	2,016	79	122	201	1,815	90.0
I. Bo	M	9	20.7	—10.7	1,996	55	102	157	1,839	92.1
H. G.	M	11	27.5	—10.7	1,979	59	96	155	1,824	92.2
J. G.	M	8	22.4	— 5.9	1,946	63	96	156	1,787	91.9
L. F.	F	10	26.9	+ 3.9	1,929	81	92	174	1,755	91.0
T. F.	M	10	23.4	— 9.8	1,854	79	93	172	1,682	90.7
I. Be	F	11	26.0	—17.2	1,838	51	109	160	1,678	91.3
R. D.	F	10	19.1	—12.3	1,819	51	75	126	1,693	93.1
M. H.	F	9	23.9	+ 3.2	1,816	72	80	152	1,664	91.6
E. F.	F	6	17.3	— 4.9	1,798	62	80	142	1,656	92.1
I. Ba.	M	10	30.5	— 4.1	1,722	55	111	166	1,556	91.4
L. C.	F	6	18.4	— 9.4	1,721	51	103	154	1,567	91.1
E. F.	F	6	19.3	+ 4.9	1,713	58	84	142	1,571	91.7
R. G.	M	6	16.3	— 7.9	1,707	48	70	118	1,589	93.1
B. G.	F	9	20.5	— 3.5	1,705	72	100	172	1,533	89.9
L. C.	F	6	18.7	— 7.9	1,691	48	127	174	1,517	93.4
E. F.	F	6	15.2	—16.5	1,680	52	99	151	1,529	91.0
A. S.	F	10	16.9	—19.2	1,675	61	71	131	1,544	92.1
E. B.	F	5	15.8	— 5.4	1,674	57	124	181	1,493	89.2
N. G.	M	7	15.0	—12.3	1,673	54	81	135	1,538	91.9
E. G.	F	6	17.5	— 1.7	1,653	53	52	105	1,548	93.7
L. P.	F	8	22.6	—19.8	1,633	57	89	146	1,487	91.1
E. G.	F	6	18.1	— 1.1	1,628	52	95	147	1,481	91.0
A. S.	F	10	17.4	—18.7	1,625	57	53	110	1,515	93.2
E. G.	F	6	17.5	— 1.7	1,620	41	96	140	1,480	91.4
A. S.	F	10	17.8	—16.8	1,608	60	67	127	1,481	92.2
L. C.	F	6	18.0	—11.3	1,604	46	87	133	1,471	91.7
R. B.	M	6	11.4	—14.1	1,587	61	100	160	1,427	89.9
N. G.	M	7	16.9	— 1.2	1,556	46	63	109	1,447	93.0
N. G.	M	7	16.5	— 3.5	1,553	50	94	144	1,409	90.7
D. Y.	F	7	17.3	—16.0	1,540	58	85	138	1,402	92.2
H. L.	F	8	19.3	—10.6	1,514	83	116	199	1,315	86.9
L. C.	F	6	17.1	—15.8	1,309	40	78	118	1,191	91.0
H. D.	M	5	13.9	+ 0.0	1,267	52	52	104	1,159	91.5
E. F.	F	6	15.4	—13.5	1,243	52	64	116	1,127	90.7
A. G.	F	8	17.9	—25.7	1,189	55	85	140	1,049	88.2
E. G.	F	6	16.3	— 8.4	1,147	44	54	98	1,049	91.5

range of from 5.7 to 9.8, according to Utheim. Since these four exceptions occur in two of the groups of undernourished children, the average of these groups is somewhat higher than for the normal group, but they still remain within the normal range.

The data given in table 6, which has been arranged in order of decreasing intake per twenty-four hours, regardless of the degree of

underweight, show that the caloric utilization per twenty-four hours is directly proportional to the intake, and also that the caloric loss per twenty-four hours, both in urine and feces, increases with the increased intake. The percentage of utilization, however, remains practically constant, and is entirely independent of the total intake. This fact is

TABLE 7.—*Caloric Balance, per Kilogram per Twenty-Four Hours*

			Weight			Output				Intake
Name	Sex	Age	Kg.	Variation from Standard	Intake	Urine	Feces	Total	Utilization	Utilization Ratio
N. G.	M	7	15.0	—12.3	111.5	3.6	5.4	9.0	102.5	1.09
E. F.	F	6	15.2	—16.5	110.5	3.4	6.5	9.9	100.6	1.04
B. G.	F	8	19.1	—13.6	108.9	3.5	4.6	8.1	100.8	1.01
E. B.	F	5	15.8	— 5.4	106.0	3.6	7.9	11.5	94.5	1.12
R. G.	M	6	16.3	— 7.9	104.7	3.0	4.3	7.3	97.5	1.07
E. F.	F	6	17.3	— 4.9	103.9	3.6	4.6	8.2	95.7	1.09
A. S.	F	10	16.9	—19.2	99.1	3.6	4.2	7.8	91.4	1.09
R. B.	M	6	16.4	—14.1	96.8	3.7	6.1	9.8	87.6	1.10
I. Bo.	M	9	20.7	—10.7	96.4	2.7	4.9	7.6	88.8	1.09
R. D.	F	10	19.1	—12.3	95.2	2.7	3.9	6.6	88.7	1.08
E. G.	F	6	17.5	— 1.7	94.5	3.0	3.0	6.0	88.5	1.07
N. G.	M	7	16.5	— 3.5	94.1	3.0	5.7	8.7	85.4	1.10
L. C.	F	6	18.4	— 9.4	93.5	2.8	5.6	8.4	85.2	1.10
A. S.	F	10	17.4	—18.7	93.4	3.3	3.0	6.3	87.1	1.07
E. G.	F	6	17.5	— 1.7	92.6	2.5	5.5	8.0	84.6	1.09
N. G.	M	7	16.9	— 1.2	92.1	2.7	3.7	6.4	85.6	1.06
H. D.	M	5	13.9	± 0.0	91.2	3.7	3.7	7.4	83.4	1.09
S. K.	M	10	25.4	—16.7	91.0	3.0	5.6	8.6	82.4	1.10
L. C.	F	6	18.7	— 7.9	90.4	2.6	6.8	9.4	81.1	1.11
A. S.	F	10	17.8	—16.8	90.3	3.4	3.8	7.2	83.2	1.09
E. G.	F	6	18.1	— 1.1	89.9	2.9	5.3	8.2	81.8	1.10
L. C.	F	6	18.0	—11.3	89.1	2.5	4.9	7.4	81.7	1.09
D. Y.	F	7	17.3	—16.0	89.0	3.1	4.9	8.0	82.1	1.08
E. F.	F	6	19.3	+ 4.9	88.8	3.0	4.4	7.4	81.4	1.09
S. K.	M	10	26.8	—12.1	87.7	2.7	4.4	7.1	80.5	1.09
J. G.	M	8	22.4	— 5.9	86.9	2.8	4.3	7.1	79.8	1.09
S. K.	M	10	27.5	— 9.8	83.9	3.0	4.2	7.1	76.7	1.09
B. G.	F	9	20.5	— 8.5	83.2	3.5	4.9	8.4	74.8	1.11
E. F.	F	6	15.4	—13.5	80.7	3.4	4.2	7.5	73.2	1.10
T. F.	M	10	23.4	— 9.8	79.2	3.4	4.0	7.4	71.9	1.10
J. U.	F	9	25.9	— 8.2	78.8	2.5	2.6	5.1	73.7	1.07
H. L.	F	8	19.3	—10.6	78.4	4.3	6.0	10.3	68.1	1.15
S. A.	F	10	29.2	+ 1.4	77.4	2.9	3.0	5.9	71.5	1.08
L. L.	M	10	34.0	+ 1.2	77.0	2.5	2.3	4.8	72.2	1.07
M. S.	M	9	33.5	+17.1	76.9	2.4	2.8	5.2	71.7	1.07
L. C.	F	6	17.1	—15.8	76.6	2.3	4.6	6.9	69.7	1.10
M. H.	F	9	23.9	+ 3.2	76.0	3.0	3.4	6.4	69.6	1.09
H. F.	M	9	26.7	— 5.8	75.9	2.3	4.0	6.3	69.6	1.09
J. M.	M	13	27.5	—12.1	75.5	2.8	3.1	5.9	69.7	1.08
H. S.	F	9	27.2	— 3.5	74.6	2.2	3.9	6.1	68.5	1.09
M. F.	F	8	27.7	+ 8.6	74.4	3.0	3.2	6.2	68.2	1.09
L. P.	F	8	22.6	—19.8	72.3	2.5	4.0	6.5	65.8	1.10
H. G.	M	11	27.5	—10.7	72.0	2.1	3.5	5.6	66.3	1.08
L. F.	F	10	26.9	+ 3.9	71.7	3.1	3.4	6.5	62.5	1.15
F. J.	F	11	28.2	+ 0.6	71.5	2.9	3.7	6.6	64.9	1.10
I. Be	F	11	26.0	—17.2	70.7	2.0	4.2	6.2	64.5	1.09
R. G.	F	10	28.6	— 3.4	70.5	2.7	4.3	7.0	63.5	1.11
E. G.	F	6	16.3	— 8.4	70.4	2.7	3.3	6.0	64.4	1.10
B. L.	F	12	33.0	+ 1.2	69.5	2.9	3.6	6.5	63.2	1.10
A. G.	F	8	17.9	—25.7	66.4	3.1	4.7	7.8	58.6	1.13
G. L.	F	13	34.2	— 3.3	63.5	1.5	3.1	4.6	58.9	1.08
I. Ba.	M	10	30.5	— 4.1	56.5	1.8	3.6	5.4	51.0	1.11

shown even more strikingly in table 7 in which the caloric exchange is expressed in terms of per kilogram of body weight. Here again the utilization is seen to be directly proportional to the intake, and the ratio of intake to utilization is almost constant. It is also interesting to note here that the urinary loss is strikingly constant when computed on the basis of body weight.

In order to determine whether there might be any relationship between the total caloric loss and the fecal weight, table 8 was arranged to show the loss of heat, caloric utilization and caloric intake per kilogram of body weight, in the order of decreasing fecal weight. It will be

TABLE 8.—*Relation of Weight of Feces to Caloric Balance*

Name	Deviation from Normal Weight Per Cent	Feces per Kg. of Body Weight Gm.	Heat Loss		Total Utilization per Kg. Calories	Caloric Intake per Kg.	Percentage Utilization
			Feces per Kg. Calories	Total per Kg. Calories			
E. B.	— 5.4	1.54	7.9	11.5	94.5	106.0	89.2
N. G.	— 3.5	1.52	5.7	8.7	85.4	94.1	90.7
L. C.	—15.8	1.37	4.6	6.9	69.7	76.6	91.0
L. C.	— 7.9	1.33	6.8	9.4	81.1	90.4	93.4
E. F.	—16.5	1.29	6.5	9.9	100.6	110.5	91.0
L. C.	— 9.4	1.21	5.6	8.4	85.2	93.5	91.1
S. K.	—16.7	1.17	5.6	8.6	82.4	91.0	90.5
E. G.	— 1.7	1.14	5.5	8.0	84.6	92.6	91.4
D. Y.	—16.0	1.08	4.9	8.0	82.1	89.0	92.2
N. G.	—12.3	1.07	5.4	9.0	102.5	111.5	91.9
E. G.	— 1.1	1.02	5.3	8.2	81.8	89.9	91.0
L. C.	—11.3	0.99	4.9	7.4	81.7	89.1	91.7
H. L.	—10.6	0.99	6.0	10.3	68.1	78.4	86.9
M. H.	+ 3.2	0.98	3.4	6.4	69.6	76.0	91.6
S. K.	—12.1	0.96	4.4	7.1	80.5	87.7	91.9
B. G.	—13.6	0.95	4.6	8.1	100.8	108.9	92.6
I. Bo.	—10.7	0.95	4.9	7.6	88.8	96.4	92.1
I. Be.	—17.2	0.93	4.2	6.2	64.5	70.7	91.3
R. G.	— 3.4	0.93	4.3	7.0	63.5	70.5	90.0
R. B.	—14.1	0.92	6.1	9.8	87.6	96.8	89.9
A. S.	—19.2	0.90	4.2	7.8	91.4	99.1	92.1
S. K.	— 9.8	0.90	4.2	7.2	76.7	83.9	91.4
F. J.	— 0.6	0.90	3.7	6.6	64.9	71.5	90.8
E. F.	— 4.9	0.88	4.6	8.2	95.7	103.9	92.1
E. F.	+ 4.9	0.88	4.4	7.4	81.4	88.8	91.7
H. F.	— 5.3	0.88	4.0	6.3	69.6	75.9	91.7
R. G.	— 7.9	0.87	4.3	7.3	97.5	104.7	93.1
E. G.	— 1.7	0.86	3.0	6.0	88.5	94.5	93.7
H. S.	— 3.5	0.81	3.9	6.1	68.5	74.6	91.8
L. F.	+ 3.9	0.80	3.4	6.5	62.5	71.7	91.0
N. G.	— 1.2	0.79	3.7	6.4	85.6	92.1	93.0
A. S.	—16.8	0.79	3.8	7.2	83.2	90.3	92.2
H. D.	± 0.0	0.77	3.7	7.4	83.4	91.2	91.5
J. G.	— 5.9	0.77	4.3	7.1	79.8	86.9	91.9
B. L.	+ 1.2	0.77	3.6	6.5	63.2	69.5	91.0
T. F.	— 9.8	0.76	4.0	7.4	71.9	79.2	90.7
H. G.	—10.7	0.72	3.5	5.6	66.3	72.0	92.2
I. Ba.	— 4.1	0.72	3.6	5.4	51.0	56.5	91.4
B. G.	— 8.5	0.70	4.9	8.4	74.8	83.2	89.9
R. D.	—12.3	0.69	3.9	6.6	88.7	95.2	93.1
M. F.	+ 8.6	0.69	3.2	6.2	68.2	74.4	91.7
G. L.	— 3.3	0.67	3.1	4.6	58.9	63.5	92.8
A. S.	—18.7	0.66	3.0	6.3	87.1	93.4	93.2
S. A.	+ 1.4	0.66	3.0	5.9	71.5	77.4	92.4
A. G.	—25.7	0.66	4.7	7.8	58.6	66.4	88.2
E. G.	— 8.4	0.63	3.3	6.0	64.4	70.4	91.5
J. M.	—12.1	0.61	3.1	5.9	69.7	75.5	92.3
L. P.	—19.8	0.61	4.0	6.5	65.8	72.3	91.1
M. S.	—17.1	0.60	2.8	5.2	71.7	76.9	93.2
E. F.	—13.5	0.59	4.2	7.5	73.2	80.7	90.7
J. U.	— 8.2	0.51	2.6	5.1	73.7	78.8	93.4
L. L.	+ 1.2	0.47	2.3	4.8	72.2	77.0	93.8

seen that while the caloric output in the feces in general decreases with the decreasing fecal weight, there is also a more or less corresponding decrease in the intake and utilization per kilogram, and the percentage utilized remains constant and entirely independent of the fecal weight or the actual loss of heat either in the feces alone or in the feces and urine combined.

CONCLUSIONS

From the foregoing data we conclude that the absorptive power of children of the degree of underweight studied by us is equal to that of the normal child. This is contrary to the conclusion reached by Utheim in her study of athreptic infants, but it is true that we were not dealing with cases of such severe emaciation as hers.

We also conclude that the absolute number of calories absorbed varies directly with the intake per twenty-four hours, and that the percentage absorbed is practically constant and may be taken as the index of normal or abnormal absorption.

In an effort to determine the use in the body of the calories absorbed, we have computed the average daily gain in weight of the children in all groups. This we may compare with the caloric intake per kilogram and the calories utilized in excess of the basal requirement. In a previous paper [9] we have reported that, referred to weight, the basal metabolism of the undernourished children was slightly higher than that of the normal group. We have already noted, in this group, that the caloric intake per kilogram of weight was also somewhat higher for the undernourished than for the normal children. We therefore find that the caloric utilization in excess of basal per kilogram of body weight was about the same for all groups, normal and underweight alike. From this we might expect to find an equal gain in weight for all groups, but, as a matter of fact, we find that at least three of the groups of underweight children gained much more rapidly than the normal group. This may be explained, however, by the fact that, as has been previously noted, the normal children were much more active than those who were underweight; therefore, a larger proportion of the calories absorbed were actually consumed, and fewer stored, by the normal than by the underweight children. The average gain in weight for group 5, the most seriously underweight of all the children, was, however, only slightly higher than that of the normal group. This is probably explained by the fact that observations were made on these children during the first week of their stay in the hospital, before they had become accustomed to the hospital routine, or had settled down to a steady gain in weight.

SUMMARY

In the study of ten normal and twenty-six undernourished children, no differences were found in the power to absorb food material or in the caloric utilization of the two groups.

The percentage absorption was the same in all groups, regardless of the degree of underweight or of the actual caloric intake.

9. Wang; Kern; Frank, and Hays (footnote 1, second reference).

The actual caloric utilization varies directly with the caloric intake, the ratio between the two being a constant with a numerical value of 1.09.

The loss of heat in the feces was found to vary in proportion to the intake, the percentage utilization remaining the same in all cases, independent of the weight of feces or the fecal heat loss.

A CLINICAL STUDY OF ENURESIS*

ADRIEN BLEYER, M.D.
ST. LOUIS

A group of children suffering with enuresis was studied; 129 were boys and 123 girls. The observation covered a period of three years. The following factors to which enuresis is commonly ascribed were selected for study:

Enlarged or diseased tonsils and adenoids.
Eye strain.
The male prepuce.
A small meatus.
Vaginitis.
Pyelitis.
Defective posture.
Malnutrition.
The neurotic constitution.
The psychic factor and the rôle of discipline.
Sex.

CAUSATIVE FACTORS IN ENURESIS

The Relation of Enlarged or Diseased Tonsils and Adenoids to Enuresis.—It was found that 116 of the children showed sufficient trouble with tonsils or adenoids to justify their classification as abnormal. In forty-nine of these, the tonsils and adenoids were removed for the purpose of curing the enuresis. This it seemed to do in one case. In one other case, the enuresis became considerably worse after the operation, and in the remaining forty-seven cases it did not seem to have any effect. This gives a rate of cure of 2 per cent for the removal of the tonsils and adenoids in enuresis.

Eye Strain.—Thirty-eight of the enuritic children suffered with defective vision or eye strain, and had either been attending or were referred to the eye service for treatment. No effect of the correction of an eye defect was observed in the members of this group.

The Male Prepuce in Relation to Enuresis.—The operation of circumcision is among the most favored remedies of enuresis in boys; it was of interest, therefore, to find that among the 129 boys, 33 had been circumcised before entering the hospital, which shows that enuresis may occur among the circumcised as well as among the uncircumcised. Indeed, fourteen of the boys had been circumcised for the relief of enuresis with a transitory effect, which lasted a few months in one case. In

* Received for publication, July 19, 1928.

* From the Department of Pediatrics, Washington University School of Medicine and the St. Louis Children's Hospital.

two instances, the parents believed that circumcision had produced the disease. Retraction of the prepuce, among the uncircumcised boys, was without benefit, except in one instance, in which the boy ceased wetting the bed at night. It was thus not possible to discover a relation between the male prepuce and enuresis in these cases.

The Relation of a Small Meatus to Enuresis.—This factor was studied in eighteen cases in which this anomaly was judged to exist. Two of the group were subjected to meatotomy and sixteen to dilatation, without any effect.

Vaginitis.—Eight of the 123 enuritic girls were suffering with a purulent vaginal discharge. This was cured in two without influencing the course of the enuresis. Four of the remaining six were cured of enuresis without attention to the vaginitis, which continued active throughout and for weeks afterward. No relation appeared to exist between the two diseases in this small group.

Pyelitis.—The coexistence of enuresis and pyelitis may be illustrated by the following cases:

CASE 1.—Enuresis was present night and day in a girl, aged 3. She was relieved of enuresis, while large amounts of pus continued to appear in her urine.

CASE 2.—A girl did not have pyelitis at the time that she was cured by massage of enuresis which occurred during the day. She subsequently acquired pyelitis, however, without a return of enuresis.

CASE 3.—A girl, in whom the administration of atropine had failed to give results, was relieved of enuresis by massage during the continuance of pyelitis. Some months later, enuresis recurred when pyelitis was not present.

CASE 4.—After two massages of the bladder, a girl was cured of enuresis which occurred during the day, without improvement in the pyelitis.

CASE 5.—Of particular interest is the case of a girl, aged 5, who had suffered all her life with diurnal and nocturnal enuresis. She had been treated at the age of 3 years for pyelitis, and, when seen two years later, the urine was found to be loaded with pus and albumin; casts were abundant. She had fever and was placed on alkalies and large amounts of water. Despite the diuretic effect of this treatment and the persistence of the pyelitis, massage of the bladder brought control for the first time in her life; the urine remained full of pus and the child continued ill.

On the other hand, that an acute pyelitis can itself cause what appears to be enuresis is illustrated by the following two cases:

CASE 6.—A girl had suffered with incessant urgency for six weeks; the urine was loaded with pus, and she had a temperature of 38.4 C. (101.1 F.), not otherwise explained. Control of micturition was acquired coincidentally with the cure of pyelitis.

CASE 7.—A child with urinary incontinence during the day was relieved by the cure of a coexisting pyelitis. It required four weeks.

These observations on the relation of pyelitis to enuresis show that among 123 girls suffering with enuresis, pyelitis was found twenty-two times or in about 18 per cent. In four of these, the enuresis disappeared with the cure of the pyelitis. In the remaining eighteen cases, the course of one disease did not seem to have the slightest effect on the course of the other disease so that, although not negligible, the relation did not appear to be one of importance. Moreover, it is desirable not to confuse pyelitis accompanied by incontinence with true enuresis.

Sex.—It is interesting to note that the number of cases of enuresis in this series is about equally divided between the sexes. This is an important fact because it suggests broadly that the cause of this disease does not reside in any anatomic or physiologic peculiarity of either sex external to the orifice of the bladder. This observation, therefore, leads one's attention away from the several foregoing items and definitely toward the bladder itself as the place where the cause of enuresis will probably be found.

Defective Posture in Relation to Enuresis.—Defective posture was observed in a small group of children suffering with enuresis, in whom a deviation from normal body mechanics was sufficiently pronounced to cause albumin in the urine when the erect position was assumed. It so happened, however, that the albuminuria in these cases did not yield to treatment during the period of observation, although it is noteworthy that the response to the treatment of enuresis in these particular children seemed to be similar to that in the general group. That is, defective posture did not seem to alter materially the course of enuresis in this series.

Malnutrition and Enuresis.—Perhaps nothing, with the exception of discipline and circumcision, has had such vogue in the treatment of enuresis as tonics, change of air or other measures for the purpose of upbuilding the body from the standpoint of nutrition and health, and for this reason a tabulation of the heights and weights of the children of this group was made. These are shown in figures 1 and 2, the lower solid line representing the normal height, the upper solid line the normal weight and the dotted lines the averages for each year of height and weight of these children. It can be seen that no inferiority appeared, such as usually accompanies malnutrition. In other words, malnutrition would not seem to have been a factor of importance in this series, and this observation does not lend weight to the time honored tonic treatment of this disease.

Enuresis and the Neurotic Constitution.—If one keeps in mind the ready response of the bladder to sensory stimuli, such as the sound of splashing water or cold on the surface of the body and the participation of the intellectual centers during emotions such as fright or excite-

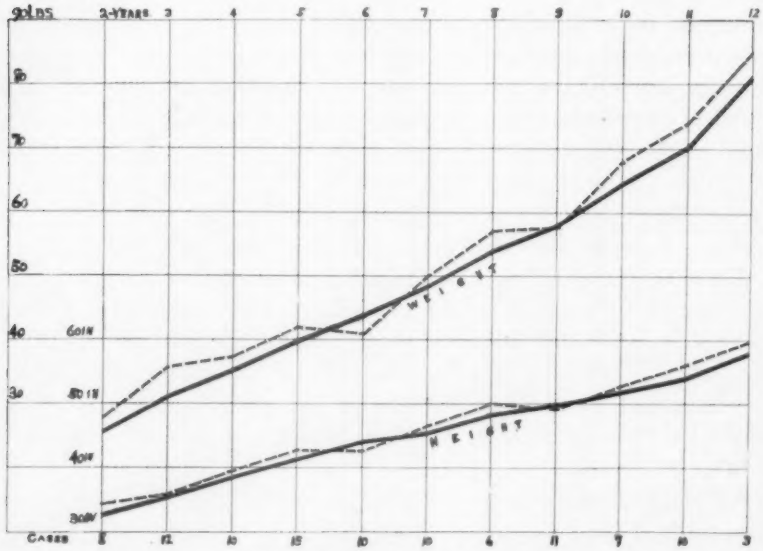

Fig. 1.—Heights and weights of the girls of the group of children studied. The lower solid line represents the normal height, the upper solid line the normal weight and the dotted lines the averages for each year of height and weight.

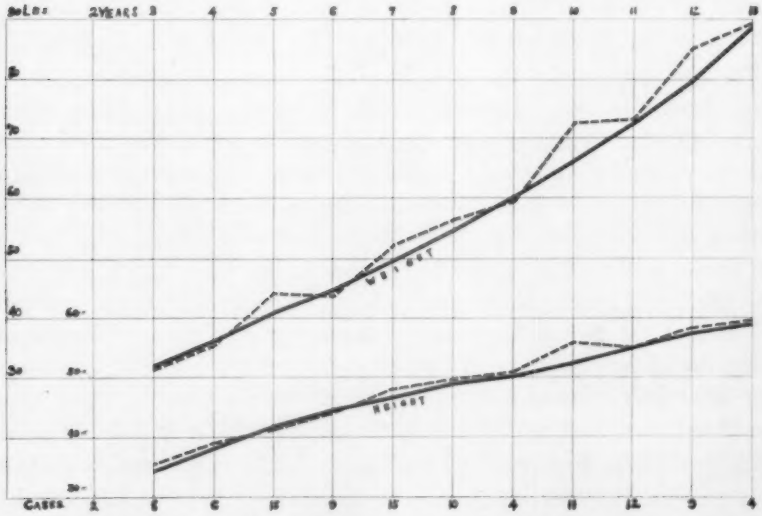

Fig. 2.—Heights and weights of the boys of the group of children studied. The lower solid line represents the normal height, the upper solid line the normal weight and the dotted lines the averages for each year of height and weight.

ment, it may be admitted that the response of the bladder will be more marked in a younger than in an older child and more marked in a child with poor inhibition at any age than in a child with inhibition centers that are more settled. Thus, less may be expected of the neurotic child of poor inhibition than of the normal child in the control of the bladder, and more may be gained in teaching control of the bladder in such a child than in the normal child through attention to the nervous system; everyone is well aware of this fact. It is this child who ceases to wet the bed during a sojourn in the hospital under cleanly and decent surroundings and who resumes it, of course, when he returns to his own home and the surroundings to which he has long been accustomed and in which the habit of indiscriminate wetting was acquired and in which he will continue to exercise it regardless of any and all treatment. It is questionable indeed whether such disturbances of the bladder of a neurotic child should be classified as enuresis or whether the cure of such a disturbance had best be called a cure of enuresis, and of this important matter another word will be said.

In the series, 25 per cent of the children seemed to show disturbances of the nervous system, evidences of what physicians are given to call instability. Some of these children were definitely defective, and the proportion was without doubt higher than is encountered in the general population of children even in dispensary practice.

A decided majority, three in four of the enuritic children of the entire series, did not exhibit the evidences of instability for which the term "neurotic" is colloquially used or in the sense in which it has been used in describing enuritic children. A large number of these children were, to the contrary, of decidedly superior type who had had the advantages of all that is best in inheritance and environment, and the plight of these children was often pathetic, because of the determined effort made to control themselves and the constant failure to do so. That such a child is wetting himself or his bed because of lack of discipline or defective will power is not true, or that he does so for the joy of urinating as was recently said to be the case by a Cleveland physician, would appear to show that some physicians are still far from agreed as to what really constitutes enuresis.

The Psychic Factor and the Rôle of Discipline in Enuresis.—Proceeding along the line that the seat of the trouble in enuresis is in the mind, a variety of expedients designed to register an impression on this faulty reasoning were tried, and for these tests the enuritic children of particularly good type mentally, nervously and physically were selected; there was an abundance of such material available. Among the hypnotic suggestions, circumcision, retraction of the prepuce and dilatation of the meatus have already been described in some detail.

The passing of ball-bougies along the canal in the region of the veru montanum, or through the sphincter was tried in a group of cases and was found to be equally useless. In twelve cases, distention of the bladder under a 75 cm. column of salt solution, as suggested by Thompson,[1] was carried out, the average number of such treatments being four for each child. No benefit was secured by this method in a single instance. Stretching of the anal sphincter was tried, because this maneuver offered a good check on the excellent effect of massage of the bladder in a goodly number of these children. None of these procedures had the slightest effect, and it would appear that they are not really worth while in the treatment of a true case of enuresis.

As to discipline in enuresis, I suppose it may be assumed that, to be effective, the bladder must be under full physiologic control of the mind. This is of course fundamental for all recourse to discipline must rest on this. The foregoing observations, however, do not appear to confirm the propriety of such an assumption, but suggest rather that, because of the ineffectiveness of such methods, it is probably this physiologic control that is sought by discipline which has been lost and that the bladder itself may actually be out of normal physiologic control. If this is true, it would seem that discipline may not be entitled to the more or less exalted position which it has occupied for a long time in the treatment of this disease. This leads to a definition of enuresis.

A Definition of Enuresis.—According to the foregoing observations, enuresis is a disturbance of micturition in which the physiologic control of the brain is blocked by stronger stimuli which have to do with the nervous mechanism of the bladder. Moreover, this loss of physiologic control does not appear to lie in the brain. This is conceived to be true enuresis, and for this type the term "enuresis vera" is proposed, separating it from another condition which, although simulating true enuresis, is a mere perversion of habits in an irresponsible, usually neurotic and often mentally defective child. To the latter condition the term "pseudo-enuresis" may be applied. The question as to whether enuresis should be classed as a true neurosis is thus opened by these observations.

THE PHYSIOLOGY OF MICTURITION AND THE TREATMENT OF ENURESIS

Certainly, the most important contribution that has been made to the study of enuresis is the discovery by Young[2] that the opening of the sphincters of the bladder is not an act of inhibition, but is brought about by the muscles of the trigone. This discovery correlates well the known

1. Thompson, A. R.: Brit. J. Child. Dis. **10**:10, 1910.
2. Young, H. H.: Surg. Gynec. Obst. **26**:608, 1918.

facts that the sphincters of the bladder, which are derived from the longitudinal and circular fibers of the detrussor and are of smooth muscle, cannot be under the control of the will and so cannot be relaxed to permit the passage of urine into the posterior urethra, whereas the muscle of the trigone, which passes through these sphincters, comprises striped muscle and is therefore capable of voluntary contraction.

Thus, it is the contraction of the trigone by the will that overcomes the tonic contraction of weaker muscles which are under the control of the involuntary system, which initiates the act of micturition, the convexity upward of the trigone at rest serving to pull the posterior wall of the orifice downward and backward as this convexity or arc disappears during contraction.

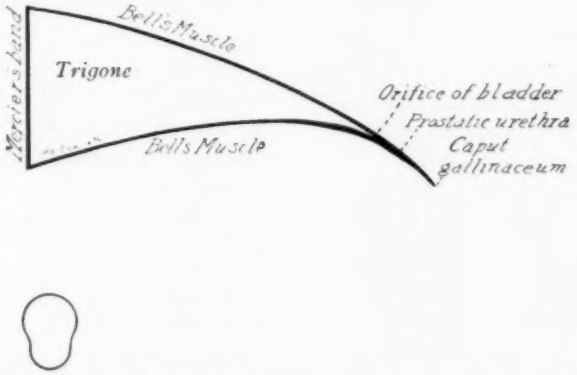

Fig. 3.—The figure at the top represents the arc of the trigone at the orifice of the bladder. The lower figure shows the posterior wall of the orifice as it is pulled downward and backward during micturition, by the contraction of the trigone. It assumes the shape of a pear. (Young.)

Since this original observation, anatomic and embryologic studies by Wesson [3] and further studies by Young [4] appear to have confirmed it. Figure 3 shows this mechanism.

The Innervation of the Bladder.—It has been difficult, it seems, to determine the physiologic response of isolated muscles of the bladder to drugs which are selective in their action on the sympathetic or parasympathetic systems because of the interlacing of fibers derived from the internal and external layers of the detrussor, which fibers pass down to form the sphincters at the orifice and also contribute to the muscles of the trigone. The studies of Macht,[5] however, seem to show that the response of the sympathetic system to epinephrine and ergo-

3. Wesson, M. B.: J. Urol. **4**:279, 1920.

4. Young, H. H.: J. Pharmacol. **12**:329, 1923.

5. Macht, D. I.: J. Pharmacol. & Exper. Therap. **8**:111, 1916.

toxine and as well the response of the parasympathetic system to pilocarpine and atropine are diametrically opposite, as between the fundus and the trigone, which may be taken to indicate that the innervation of the trigone is more or less independent of that of the rest of the bladder.

Autonomic fibers reach the bladder through the vesical plexus from the first, second and third sacral segments, and by virtue of their ganglions are engaged in some phase of micturition. There is not any peristaltic action, however, in the bladder beyond the orifices of the ureters, and this does not lend support to the view that enuresis may be related to an important degree to the parasympathetic system. The sympathetic system is represented in the bladder by filaments from the third, fourth and fifth lumbar nerves, and it may be somewhat more probable that these play a part of great importance in the disturbed mechanism of the bladder in enuresis.

Until the physiologists can clear this point, it may be impossible for clinicians to acquire satisfactory knowledge of the mechanism at fault or to explain what appears to be the crux of the matter, namely, that the child suffering with enuresis is actually urinating before he wants to do so and that this is due to the fact that urine has been allowed to reach the prostatic urethra through local nervous reactions which for the time are not subject to the control of the will. The compressor urethrae, composed of voluntary muscle, comes into action to complete the act of micturition, but remains passive throughout its earlier phase.

The Action of Atropine in Enuresis.—Atropine is a specific remedy for enuresis, and failure to effect a cure with it does not indicate that this is not true but rather that physicians are not in possession of all the facts that are needed to understand why failure occurred. In the series, success with atropine was noted in about 80 per cent of the patients treated, which corresponds with a report by Ostheimer and Levi,[6] in Griffith's service some years ago. It is necessary in the use of atropine, for the cure of enuresis, to get the dosage as near the margin of overdosage (flushing, mydriasis) as is possible, and in this study a solution of 0.25 grain (0.016 Gm.) to the ounce rather than 1 grain (0.06 Gm.) to the ounce was used, because a closer adjustment of the drug to the individual need could in this way be secured, each drop representing about 0.002 grain instead of 0.05 grain. There is little danger in the use of atropine, if used with ordinary care.

Massage of the Bladder in Enuresis.—Massage of the bladder is carried out by inserting the finger into the rectum and touching the trigone. The bladder can be brought against the pubes without pain

6. Ostheimer, Maurice and Levi, I. V.: Enuresis in Childhood, J. A. M. A. **43**:1844 (Dec. 17) 1904.

to the patient, but this was not found to add to the effectiveness of the procedure; the pressure should be light and need not be continued for more than a moment. Its effect is noticed almost if not at the beginning of the treatment, and if found ineffective after the third or fourth trial, it should be given up. Its effect seems to be more or less limited to the patients with enuresis during the day, in whom urgency is the chief symptom and in cases in which the child, who must urinate every few minutes, is never relieved of the desire to urinate. Patients with nocturnal enuresis do not, as a rule, respond to massage. The duration of the enuresis does not materially influence the effectiveness of the treatment. It is good practice to try the effect of massage a few times, at intervals of one or two days, in a patient with enuresis during the day, before atropine is prescribed. And it is well to bear in mind that there is no mental or suggestive part in this procedure, and for practical reasons it is advisable that the idea that there is be discarded altogether during the treatment of the disease.

CONCLUSIONS

1. Clinical analysis of certain factors commonly held accountable for enuresis failed to show their relation to this disease.

2. Cures in this series of cases amounted to about 80 per cent and were accomplished without regard to any of these factors by measures directed to the bladder itself.

3. A study of the incidence of enuresis in relation to sex suggests that the cause of this disease does not reside in any anatomic or physiologic peculiarity external to the orifice of the bladder.

4. The rôle of the voluntary nervous system is unimportant in enuresis. This disease is, in effect, a disturbance of the physiology of micturition, probably due to stimuli arising in the bladder itself which for the time being place it beyond the control of the will. Discipline plays no part in true enuresis, since this disease does not owe its origin to a lack of discipline nor is it controlled by this measure.

5. In conformity with the definition of enuresis evolved in this study, the term "enuresis vera" is suggested to indicate that type in which dissociation between the brain and the voluntary nervous system, as concerns the physiology of micturition, actually appears to exist, and the term "pseudo-enuresis" is reserved for that type of defective control which can be relieved by measures designed to influence the will or to obtain the cooperation of the child.

6. Atropine and massage of the bladder were the only measures found to be effective in the treatment of true enuresis.

University Club Building.

Case Reports

SICKLE CELL ANEMIA*

MARTHA WOLLSTEIN, M.D.
AND
KATHERINE V. KREIDEL, A.B.
NEW YORK

At the Babies' Hospital, interest in sickle cell anemia dates from a case seen in December, 1925.

REPORT OF CASES

CASE 1.—A colored girl, aged 3 years and 11 months, was admitted to the hospital with pain and stiffness in the joints of both elbows. An examination of the blood revealed the fact that 12 per cent of the red cells were sickle-shaped. She had been born at term, weighing 6 pounds (2.7 Kg.), and was first seen in the outpatient department at the age of 9 months, with the complaint of cough and sweating. When seen again at the age of 3 years, she was anemic; the spleen was hard and palpable, and the liver reached the costal margin. There was a yellowish-green discoloration of the sclerae, and the cervical glands were enlarged. Three months later, she complained of abdominal pain and pain in both arms, and was admitted to the hospital, where she remained under observation for one month. X-ray examination of the extremities did not reveal a lesion, and the pain subsided in a week. The spleen was not palpable. This patient has been followed at frequent intervals to the present time. Table 1 shows the results of the examination of the blood. The hemoglobin has remained under 40 and has been as low as 25. The red cells diminished from 3,000,000 to 1,600,000, whereas the platelets were always normal. The leukocytes were consistently high, between 12,700 and 26,000, the polymorphonuclears varying from 72 to 43 per cent. The highest number of normoblasts was 9 per hundred leukocytes. Reticulocytes were always increased, the highest number being 15 per five hundred cells counted. In the stained smear, sickling as high as 16 per cent was present, and after incubation 100 per cent of the red cells underwent the change from round to sickle shape. The patient was an only child, and examination of the parents showed that the father's blood was normal, while the mother's blood, on incubation, showed 75 per cent of sickling. Wassermann tests on parents and child were negative. Fragility tests on the child's red cells gave a normal range. The icteric index was 18.7 on one day and 21.4 on another. The van den Bergh test gave a positive, delayed, direct reaction, indicating increased hemolysis. The urine was negative for albumin and casts, and its urobilin content was positive in the dilution of 1:40. The chemical analysis of the blood showed nothing abnormal. At the age of 6, the patient still had recurrent attacks of abdominal pain and vomiting, with a persistent cough. She was of average size and intelligence, but extremely anemic, with a constant, yellowish-green discoloration of the sclerae. At the time of writing, the spleen was distinctly enlarged, and the liver was palpable. This variability in the size of the spleen was also noted in case 3.

* Received for publication, June 19, 1928.

* From the Babies Hospital and the Department of Diseases of Children, College of Physicians and Surgeons, Columbia University.

The statement has been made by Steinfeld and Klauber [1] and by Jaffé [2] that patients with sickle cell anemia do not die of the anemia itself, but of some complicating disease. Our experience with two children, who entered the hospital in the same week, led us to believe that there are exceptions to this rule, and that patients with sickle cell anemia may die as a result of the anemia without any other cause of death demonstrable at autopsy. These two cases, both fatal within a few hours after the patients entered the hospital, when so rapid a fatal outcome had not been anticipated from the history or from the condition on admission, directed our attention anew to the subject of sickle cell anemia.

TABLE 1.—*Blood Counts in Case 1, from Oct. 21, 1925, to April 9, 1928*

Date	Hemoglobin	Red Blood Cells	Platelets	White Blood Cells	Polymorphonuclears	Lymphocytes	Monocytes	Eosinophils	Reticulocytes	Normoblasts	
10/21/25	55	3,408,000	.....	15,300	40	45	15	..	....	..	
12/17/25	45	2,208,000	176,000	17,350	54	38	6	2	12.0	1	1 megaloblast
12/19/25	40	2,576,000	192,000	17,850	77	27	1	6	24.0	9	
12/28/25	40	3,000,000	352,000	17,400	50	46	3	1	....	2	
1/ 7/26	35	2,336,000	368,000	21,300	72	20	7	1	....	8	
1/12/26	30	2,224,000	264,000	26,350	64	28	4	2	....	1	
5/23/26	40	2,368,000	136,000	17,100	39	56	4	1	....	1	
6/ 7/26	35	1,768,000	176,000	21,700	49	48	1	2	....	4	
7/ 2/26	40	2,464,000	288,000	17,500	54	42	3	1	11.0	..	
8/ 4/26	30	1,856,000	240,000	16,800	50	38	1	1	12.0	..	
9/29/26	45	2,800,000	264,000	17,450	13	41	6	..	4.6	1	
1/ 3/27	35	2,408,000	288,000	18,850	43	50	1	3	15.0	2	1 myelocyte
2/ 4/27	35	2,304,000	224,000	19,150	58	38	3	..	....	4	
3/11/27	30	1,808,000	320,000	12,700	53	40	6	1	....	..	
5/ 4/27	30	1,848,000	328,000	15,250	52	43	3	2	8.2	3	
6/16/27	25	1,616,000	.....	21,200	57	39	3	1	....	..	
2/ 6/28	35	1,864,000	344,000	14,100	46	50	2	..	....	1	1 basophil, 1 myelocyte
4/ 9/28	30	2,352,000	392,000	15,300	82	17	1	..	....	2	

CASE 2.—*History.*—A colored girl, aged 4 years, was admitted on Feb. 6, 1928. with a diagnosis of acute rheumatic fever and acquired heart disease with decompensation. She was the third of five children and was born after a normal delivery, weighing 5½ pounds (2.6 Kg.).

When 6 months old she had had convulsions, and since that time had had attacks of vomiting and epigastric pain every other month. Since the age of 2 years she had repeatedly complained of pain in the knees and ankles, more marked during the gastric attacks. A sore throat had developed three weeks before examination. On admission, she was fairly well nourished, acutely ill, dyspneic and extremely anemic. The heart was enlarged, rapid and regular. A mitral systolic murmur was heard. The liver extended 5 cm. below the costal

1. Steinfeld, E., and Klauber, J. V.: Sickle Cell Anemia, M. Clin. N. Amer. **10**:1561, 1927.

2. Jaffé, R. H.: Die Sichelzellenanämie, Virchows Arch. f. path. Anat. **265**: 452, 1927.

margin, but the spleen was not palpable. The temperature was 98.6 F. During her stay in the hospital the child was drowsy and moderately prostrated, with an anxious expression, marked dyspnea and periods of restlessness. She was too weak to cooperate when examined, and appeared to have pain in the abdomen. She died quietly eighteen hours after admission.

Autopsy.—The body was that of an emaciated, female, colored child. There were no skin lesions. Examination of the head was not permitted. The upper lobe of the left lung was well aerated, with emphysema along the anterior border and in the lingula. In the posterior portion of the lower lobe of the left lung there was a wedge-shaped area of organized pneumonia 4 cm. high, 1 cm. wide and 1 cm. deep. Between the upper and lower lobes there were organized adhesions. The lower lobe of the right lung was adherent over its posterior border, and there were many adhesions between the lobes. The upper lobe of the right lung was aerated and emphysematous along its anterior border. The middle lobe, which was not well differentiated from the upper lobe, contained an area of organized

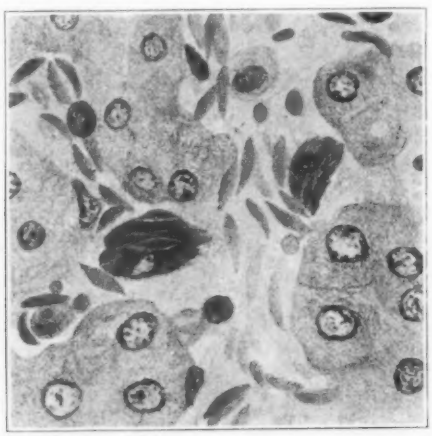

Fig. 1 (case 2).—Section of the liver. The sinusoids are wide; almost all the red cells are sickle-shaped, the Kupffer cells are large, and contain sickle cells, pressing the nucleus to one side.

pneumonia 4 by 2 by 1 cm. Over this, the pleura was gray and thick. The tracheobronchial lymph nodes were enlarged only at the bifurcation of the trachea, where they were moist and pale. The thymus gland weighed 15 Gm.; the left lobe was larger than the right. The heart measured 6 cm. vertically and 6.5 cm. transversely. The pericardium was normal; and the myocardium was pale and flabby. The wall of the left ventricle was about three times as thick as the wall of the right ventricle. All the valves were normal. The foramen ovale and the ductus arteriosus were closed. The spleen weighed 30 Gm. and was very firm and hard. The capsule was irregularly thickened along the inner surface, and on section the dark red pulp was firm and smooth. The liver weighed 460 Gm. It was firm, moderately fatty and congested. The gallbladder and ducts were normal. In the wall of the upper part of the jejunum there was a small, round nodule which proved to be a cyst 1 by 0.5 cm. in diameter. In the lower portion of the ileum, the solitary follicles were distinctly enlarged. The colon was normal throughout. The mesenteric nodes were large and pale, measuring

from 0.5 to 2 cm. in length. The kidneys were firm and red. The capsules were free, the surface smooth and the cortex swollen.

Anatomic Diagnosis.—The diagnosis was chronic pleurisy, chronic pneumonia, pulmonary emphysema, myocarditis, general adenopathy, fatty degeneration of the liver, congestion of the spleen, chronic perisplenitis, parenchymatous degeneration of the kidneys, cyst of the intestinal wall and icteric staining of the sclerae.

Bacteriology.—Culture from the bronchial secretion showed *Staphylococcus citreus* and *Streptococcus hemolyticus.* The culture of blood from the heart did not give any growth.

Microscopic Examination.—The sinusoids of the liver were wide, but contained few blood cells. The Kupffer cells were large and had phagocytosed many sickle-shaped red cells (fig. 1). The red cells in the sinusoids were almost all sickle shaped, and in the larger veins the same was true. The cells of the liver were granular and the majority contained drops of fat. The bile capillaries were

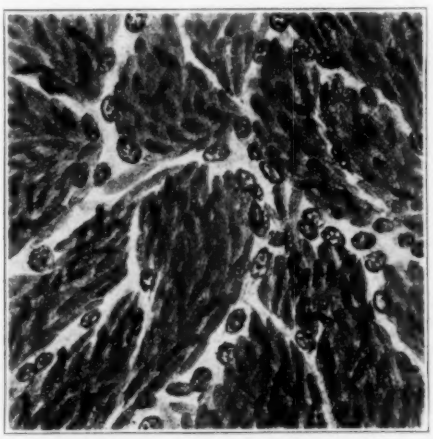

Fig. 2 (case 2).—Section of the spleen. The sinusoids are distended with sickle-shaped red cells.

empty and the bile ducts were normal. The connective tissue was normal in amount. Granules of pigment were present, and the stain for iron (potassium ferrocyanide, Mallory) gave a positive prussian blue reaction in the Kupffer cells. The distention of the sinuses of the spleen was intense, and the red cells in them were almost entirely sickle-shaped. In the less densely filled sinuses, some normal red cells and granules of brown pigment were visible, and the endothelial cells were distinct. Hemorrhages were not present. The septums of connective tissue were not thickened. The malpighian bodies were small, evidently compressed and without germ centers. The capsule was normal and its covering cells were intact. The impression was one of masses of sickle-shaped red cells in the sinuses of the spleen, where they seemed to whorl around (fig. 2). The potassium ferrocyanide stain showed much prussian blue precipitate in the endothelial cells, but not in the septums. The congestion of the capillaries and of the larger vessels of the lungs was marked, the red cells being sickle-shaped. Some alveoli were empty; some contained coagulated serum, and some contained red blood cells with a few mononuclears. There were lobules in the lungs, in which the alveolar walls were

in contact and the lining cells distinct, while other alveoli contained plugs of connective tissue. Frozen sections of the heart, stained with osmic acid and others stained with sudan III, showed the presence of fat globules in some of the muscle cells, irregularly distributed (fig. 3). Paraffin sections showed a mild degree of fatty change in the muscle cells, but did not show interstitial infiltration. The vessels were almost empty, but sickle cells and a few leukocytes were present. The capillaries of the glomeruli of the kidney were distended with sickled red cells, and the glomeruli were large. Neither exudation nor proliferation was apparent. The epitheliums of the convoluted tubules showed a large amount of degeneration and peeling so that the tubes were wide, and granular casts were present in Henle's loops as well as in the collecting tubes. Hemosiderin pigment was present in the epitheliums of the convoluted tubules, Henle's loops and the collecting tubules. The connective tissue of the kidney was normal and all the larger vessels were filled with red cells. Intact epithelium was present over a papilla and its calyx wall, which was normal. The lymph nodes and the cords were densely packed

Fig. 3 (case 2).—Frozen section of the myocardium, stained with osmic acid. Fat droplets are noted in the muscle cells.

with lymphocytes, and the sinuses were almost empty. Phagocytosis was not marked, nor was the connective tissue thickened.

Before case 2 had been studied microscopically, another colored child died seven hours after admission to the hospital.

CASE 3.—*History.*—A boy, aged 3 years 4 months, had been seen in the dispensary at the age of 3 months, with eczema. At that time, the abdomen was normal. When 2 years old, he had had an attack of tonsillitis with swelling of the cervical glands. About nine days before admission, the child had complained of abdominal pain, but had played about as usual during the following week. Then the pain had returned, and he had vomited several times. The night before he was brought to the hospital the mother had noticed that the boy "had a lump in the abdomen under the rib." On admission, the liver was palpable, there was a general glandular swelling and the spleen was markedly enlarged. The sclerae were geenish yellow, and for this reason a drop of blood was placed under a cover glass ringed

with petrolatum to be studied for sickling. The temperature on admission was 100 F., but the child seemed acutely ill. The heart was not enlarged. A loud murmur was present at the base of the heart and transmitted upward to the axilla. At the base of the right lung the breath sounds were diminished and occasional râles were heard. The patient was dyspneic, and died quietly and unexpectedly seven hours after admission. The blood showed 100 per cent sickle cells after standing over night. The red blood cells of his mother and of his two sisters showed sickling after standing.

Autopsy.—The body was that of a poorly nourished, male, colored child. Rachitic enlargements were not present. The abdomen was flat, but the spleen could be palpated. No skin lesions were present. The conjunctivae were mildly bile-stained. Examination of the brain was not permitted. The lungs were well aerated, with many subpleural hemorrhages, 1 and 2 mm. in diameter, and a moderate edema. The larynx, trachea and bronchi were normal. All the bronchial lymph nodes were enlarged, moist and congested. At the tracheal bifurcation the longest measured 1.5 cm. The pericardial sac contained a slight excess of clear serum, so that it measured 7.8 by 7 cm. The myocardium was pale and flabby. The foramen ovale and the ductus arteriosus were closed. The thymus weighed 18 Gm. It was pink and wide, but not thick. The spleen weighed 210 Gm., and was dark blue and exceedingly firm. The capsule showed areas of keratosis, but the septums were not thickened. In the deeply congested pulp, some normal-sized malpighian bodies were present. The liver extended 7 cm. below the sternal notch and 6 cm. below the ribs in the right mammary line. It weighed 460 Gm. and was congested, fatty and firm. The gallbladder and ducts were normal. The stomach was normal. In the ileum and throughout the colon, the solitary follicles were enlarged, and were more than usually numerous. The mesenteric lymph nodes were enlarged and pale; the largest measured 2 by 1 cm. The kidneys weighed 114 Gm. They were pale; the capsule stripped readily and the cortex was swollen.

Anatomic Diagnosis.—Hyperplasia and congestion of the spleen, pulmonary edema, subpleural hemorrhages, general adenopathy, icteric staining of the sclerae, congestion and fatty degeneration of the liver and parenchymatous degeneration of the kidney were diagnosed.

Microscopic Examination.—Many of the alveoli of the lungs were empty; others contained coagulated serum and a few mononuclear cells. The capillaries and the larger vessels were filled with red blood cells, many of which were sickled. The sinusoids of the liver were wide, but they contained few red cells. The Kupffer cells were large, and were filled with sickle cells. The liver cells were granular and some contained small drops of fat, but most of the nuclei still stained well. The bile ducts and the connective tissue were normal. Iron pigment was present in the Kupffer cells. The section of the spleen gave the appearance of a mass of blood with a few malpighian bodies showing germ centers. With the high power lens, it was apparent that the sinuses were distended with red cells which were all of the sickle type. Hemorrhages into the pulp had not occurred, and the blood was contained within the widely distended sinuses, the lining cells of which were distinct and not proliferating. The connective tissue septums of the spleen were not increased in thickness, and the capsule was normal, with its covering cells intact. There was no iron pigment in the trabeculae, but the phagocytic cells in the sinuses did contain some pigment. The difference between our cases and Jaffe's,[2] on this point, may indicate a difference in age of the lesion. The absence of calcium in the trabeculae would bear this out. Foot

demonstrated reticulum thickening in the case reported by Bell and his associates. The follicles of a lymph node showed large germinal centers and the rest of the node was densely packed with lymphocytes, so that the cords were largely obliterated. The small veins were filled with red sickle cells. Frozen sections of the heart stained with sudan III showed fat in many of the cells. The interstitial tissue was normal. The veins were filled with red cells which showed sickling. The endocardium was normal. There was a large amount of epithelial swelling in the convoluted tubules of the kidneys, so that many of these contained granular material which was also present in Henle's loops. Iron pigment was found in the epitheliums of the convoluted tubules, Henle's loops and the collecting tubules (fig. 4). The glomerular capillaries contained red cells and the subcapsular space contained granular material. No inflammatory lesion was present. The blood cells within the capillaries and the larger veins were sickled. The connective tissue was normal. The wall of a calyx was normal, with its lining cells intact. The periphery of the lobules of the thymus gland was densely packed with lympho-

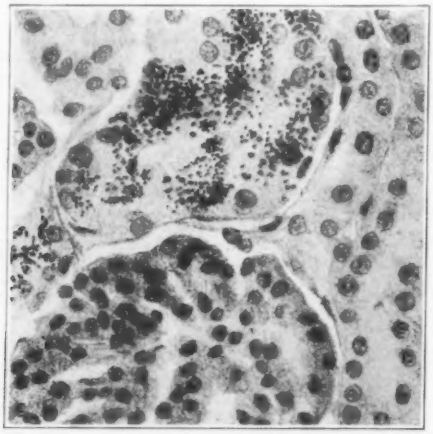

Fig. 4 (case 3).—Section of the kidney, stained with potassium ferrocyanide (Mallory). Iron pigment (prussian blue precipitate) is noted in the epithelial cells of the tubules.

cytes, while the central portion was less dense, with numerous Hassall bodies, some of which contained polymorphonuclear leukocytes. Some large reticulum cells contained pigment granules.

Looking over our autopsy records on negro children during the last six years, we found one which agreed with those reported.

CASE 4.—*History.*—A boy, a West Indian negro, aged 3 years, normal at birth and breast fed for nine months, had always been "a weakly child." Two days before entering the hospital he had been fretful and feverish, had vomited and had bled from the nose, as he had often done before. On admission, there was a general adenopathy; the liver was enlarged, but the spleen was not palpable. Five hours after admission, he died.

---

3. Bell, A. J.; Kotte, R. H.; Mitchell, A. G.; Cooley, T. B., and Lee, P.: Sickle Cell Anemia; Two Cases with Splenectomy, Am. J. Dis. Child. **34**:923 (Dec.) 1927.

Autopsy.—The body was that of a well developed and well nourished, male, colored child. No skin lesions were present. The lungs were well aerated, pink and congested. The bronchial nodes were not enlarged. The pericardial sac was slightly dilated with clear fluid. The heart was larger than normal, but the valves did not show a lesion. The muscle was pale. The thymus extended down on the pericardium beyond the fourth rib. It was soft and weighed 18 Gm. The spleen extended 4 cm. below the costal margin and was large and hard, and weighed 182 Gm. The capsule was thickened on the inner surface. The substance was firm and congested, and the malpighian bodies were not visible. The liver was firm and smooth, weighing 430 Gm. The kidneys were red and lobulated. The markings were blurred and the cortex was thickened. Examination of the brain was not permitted. Culture of the heart's blood did not show any growth.

Microscopic examination showed changes in the liver, spleen and kidneys similar to those in cases 2 and 3.

In none of these three cases was any acute cause for death demonstrable by gross or histologic study of the organs. Cultures of the blood at autopsy remained sterile. The presence of hemolytic streptococci in the bronchi in case 2 was not accompanied by any inflammatory reaction. Whether they were in the bronchi before death is a question. The possibility that their presence in the upper respiratory tract contributed to the unexpected death is a remote one. The fatty degeneration in the heart and liver, the parenchymatous degeneration of the liver and kidneys, the subpleural hemorrhages in case 3 and the general adenopathy are explained by the anemia. The splenic enlargement was due chiefly to distention of the sinuses with sickle cells, and gave evidence of the increased work required of the spleen to care for these enormous numbers of abnormal cells.

Microscopic examinaion of the sections from these cases demonstrated their similarity in: the presence of sickle cells in the blood vessels in all the organs, the phagocytosis of sickle cells by Kupffer cells in the liver and the presence of iron pigment in the Kupffer cells and in the tubules of the kidney as shown by the potassium ferrocyanide reaction. In two cases, the heart was slightly enlarged; the myocardium was pale and flabby and in frozen sections, stained with sudan III or osmic acid, fatty degeneration was shown. As bones could not be removed, only smears of marrow were available. These showed many nucleated red cells, many sickle cells and some nucleated sickles. The spleen in the second and third cases was much larger than in the first, but the microscopic picture was practically the same. The liver was much enlarged, and the characteristic histologic picture was one of phagocytosis of the sickle cells by the Kupffer cells and the presence of iron in these cells, but not in the liver cells, which were granular and contained small drops of fat. In sections from every organ, sickle cells were present in the capillaries and in the larger vessels. Marked lesions were not present in the lungs in any case, although in case 2 a wedge

of organized pneumonia was present in one lobe. This was neither severe enough nor extensive enough to cause death, but was the result of a previous respiratory infection, to which these children are distinctly prone.

In addition to these three patients, who died of anemia without any other anatomic cause for death, the hospital records contain an account of seven autopsies performed on colored children sections of whose organs showed marked sickling of the red cells. From the histories it is apparent that four were active cases of sickle cell anemia and three were in the latent stage; two of these patients (aged 7 and 4 months, respectively) died of miliary tuberculosis, and the third, a boy, aged 14 months, who had been under observation for congenital syphilis, died of pneumococcus meningitis. One of the patients with active sickle cell anemia also died of pneumococcus meningitis. Two other patients with active sickle cell anemia died of bronchopneumonia at the age of 3 months and 13 months, and one died of tetany after an illness of only one day. Besides these ten cases which came to autopsy, we are following five children from 1 to 12 years of age with sickle cell anemia who have been under observation for a period varying from one month to two and one half years. Among our fifteen cases, there are seven girls and eight boys, varying from 3 months to 12 years of age.

There are certain clinical symptoms common to all the active cases: discoloration of the sclerae; hemoglobin under 50 per cent; sickle cells in the smears; moderate leukocytosis; urobilin in the urine; increased icteric index; indirect, delayed van den Bergh reaction; normal fragility of the red cells, without increased hemolysis on standing at room or body temperature. Table 2 gives the blood counts in eleven cases. A history of attacks of vomiting, abdominal pain or pains in the joints, a systolic murmur and a slight enlargement of the heart are present in most cases. General, mild adenopathy is usually present. The ulcers of the leg described in older patients were not encountered in the young children. In our series, the spleen was palpable in all but two, and these were both in the active phase. According to Miyamoto and Korb,[4] the spleen is almost always small. Sydenstricker[5] found the spleen palpable in eleven of seventy-one patients and then only in the presence of malaria. In Alden's[6] patient, aged 4, the spleen was just palpable.

---

4. Miyamoto, K., and Korb, J. H.: Meniscocytosis (Latent Sickle Cell Anemia): Its Incidence in St. Louis, South. M. J. **20**:1912, 1927.

5. Sydenstricker, V. P.: Sickle Cell Anemia, South. M. J. **17**:177, 1927.

6. Alden, H. S.: Sickle Cell Anemia: Two Cases from Ohio Illustrating Its Hemolytic Nature, Am. J. M. Sc. **173**:168, 1927.

Hahn and Gillespie [7] found the spleen at the crest of the ileum, in a boy of 4 years, and the following year Hahn [8] reported the case of a boy 18 months old, whose spleen was four times the normal size. In Stewart's [9] patient, the spleen was large at the age of 16 months and at 2 years, it was not palpable at 4 years, and weighed 46 Gm. when removed at the age of 5 years 8 months. The spleen in Bell's [3] patient, 19 months old, extended to the iliac crest and weighed 186 Gm. on removal. In Cooley's [10] case, the spleen weighed 194 Gm. in a girl 2½ years old. Among our ten fatal cases, the spleen had been palpable before death in eight and not palpable in two. In the three children who died from anemia, the spleen weighed 30, 210 and 182 Gm., the children being 4, 3 and 3 years old. The spleen was not enlarged in two infants, 3 months and 13 months old, who died of bronchopneumonia. A girl, 2 years 11 months of age, who died of pneumococcus meningitis, had a spleen weighing 102 Gm., and in a boy 1 year of age, who died of tetany, the spleen weighed 20 Gm. A child with congenital syphilis and latent sickling, who died of pneumococcus meningitis when 14 months old, had a spleen of normal size. In two other patients with latent forms, who died of tuberculosis, the spleen was enlarged (36 Gm.) in one who lived four months, and not enlarged in the other who was 7 months of age.

While it is evident that the spleen was markedly enlarged in three active cases, in two of which the patients died of anemia and in one of pneumococcus meningitis, the spleen was moderately enlarged in one other patient, who died of pneumonia, while in three cases it was of normal weight for the child's age. Of three with latent cases, the spleen was moderately enlarged in only one patient; this patient died of general miliary tuberculosis. In this series, no child had a spleen smaller than normal. In both of Jaffé's [2] patients, 6 and 8 years old, the spleen weighed only 10 Gm. In the case described by Sydenstricker, Mulherin and Houseal,[11] a boy, aged 5 years, had a spleen that weighed 7.9 Gm. That the spleen varies in size at different times is evidenced by two of the cases in this series, and by Stewart's [9] case.

---

7. Hahn, E. V., and Gillespie, E. B.: Sickle Cell Anemia: Splenectomy, Arch. Int. Med. **39**:233 (Feb.) 1927.

8. Hahn, E. V.: Drepanocytic Anemia, Am. J. M. Sc. **175**:206, 1928.

9. Stewart, W. B.: Sickle Cell Anemia: Report of a Case with Splenectomy, Am. J. Dis. Child. **34**:72 (July) 1927.

10. Cooley, T. B., and Lee, Pearl: The Sickle Cell Phenomenon, Am. J. Dis. Child. **32**:334 (Sept.) 1926.

11. Sydenstricker, V. P.; Mulherin, W. A., and Houseal, R. W.: Sickle Cell Anemia: Report of Two Cases in Children, with Necropsy in One Case, Am. J. Dis. Child. **26**:132 (Aug.) 1923.

It would seem that in patients with sickle cell anemia in the first four years of life the spleen varies in size, probably enlarging with exacerbations of the anemia, whether these are due to acute infections or to a factor which has not yet been demonstrated. At autopsy, the spleen in these young children may be markedly enlarged, moderately enlarged or of normal size. In older children, the spleen would seem to grow smaller, possibly as the result of hemorrhage and fibrosis. That infants with latent sickling of the red cells do not always respond to severe infections with marked splenic enlargement is evidenced by the 14

TABLE 2.—*Blood Counts in Eleven Cases of Sickle Cell Anemia*

Name	Age	Date	Hemoglobin	Red Blood Cells	Platelets	White Blood Cells	Polymorphonuclears	Lymphocytes	Monocytes	Eosinophils	Reticulocytes	Normoblasts	
M. E.,	6 mo.,	1/19/22	..	.....	.....	8,100	64	32	5	..	...	..	
		1/25/22	..	.....	.....	7,650	33	67	..	..	...	..	
R. T.,	3 mo.,	7/25/25	65	.....	.....	9,000	80	10	7	..	...	..	Myeloblast 1
J. D.,	8 mo.,	11/19/25	80	4,912,000	344,000	16,950	78	22	..	..	...	6	
L. G.,	13 mo.,	2/24/26	40	4,208,000	.....	7,800	37	60	3	..	...	..	Megaloblast 1
G. P.,	4 yr.,	3/11/26	30	3,320,000	.....	14,000	60	35	4	..	...	..	Myelocyte 1, basophil 1
		3/12/26	30	3,504,000	208,000	6,350	55	37	5	..	6.0	..	Myelocyte 1, basophil 2
		3/16/26	30	3,976,000	286,000	14,000	66	28	4	..	0.2	..	Myelocyte 1, basophil 1
G. S.,	10 yr.,	7/ 1/26	40	2,576,000	.....	13,700	53	29	18	..	...	..	
		6/25/27	40	2,848,000	.....	10,800	48	47	3	1	...	..	
H. S.,	5 mo.,	5/16/27	50	.....	.....	6,600	55	42	2	1	...	..	
		6/17/27	50	3,648,000	.....	8,100	35	59	2	2	...	..	
		11/ 4/27	50	.....	.....	5,950	39	61	..	..	...	..	
G. F.,	1 yr.,	6/15/27	50	.....	.....	16,200	78	22	..	..	...	..	
		6/20/27	45	3,536,000	672,000	18,700	50	44	6	..	0.8	..	
N. G.,	3 yr.,	2/ 9/28	20	1,808,000	.....	14,450	69	29	2	..	...	..	
V. A.,	2 yr.,	2/25/28	40	2,472,000	128,000	19,750	90	7	2	..	5.2	..	
H. N.,	5 yr.,	2/18/28	35	1,816,000	544,000	14,150	81	19	..	..	...	..	
		3/ 5/28	25	1,768,000	432,000	15,800	70	20	1	..	1.0	1	Basophil 1
		3/21/28	35	2,552,000	480,000	18,300	74	20	4	2	4.0	..	
		4/ 2/28	35	2,612,000	368,000	24,400	72	26	1	1	3.4	5	

months old boy who died of pneumococcus meningitis, whose spleen was of normal size at autopsy.

The liver was enlarged in nine of the fifteen cases, extending from 1 to 5 cm. below the costal margin. It was not enlarged in one case, an inactive one, and was felt at the costal margin in five instances. At autopsy, it was distinctly enlarged in seven cases, and not enlarged in three, one of which was inactive.

The heart was stained for fat in three active cases and droplets were demonstrated in the myocardial cells. Interstitial reaction did not occur.

The thymus gland was not enlarged in any of our cases. The solitary follicles of the intestines were hyperplastic in case 2.

The marrow showed marked activity in its attempt to replace the hemolyzed sickle cells, and thus young cells (reticulated and nucleated) were present in the circulating blood.

The pathogenesis of the condition is still obscure. It occurs only in negroes, full blooded or mixed. Stewart's [9] patient, though apparently white, was of Cuban descent. The subject of elliptic human red blood cells in white persons, which has a familial incidence, has been reviewed by Huck,[12] who found it not related to sickle cell anemia. Lawrence's [13] patients belong in this group. Castana's [14] case is a doubtful one, and that recently reported by Bernhart [15] can hardly be accepted as a proved case of sickle cell anemia in a white person, in the absence of details and illustrations. Emmel [16] first demonstrated the familial occurrence of sickle cell anemia. In the outpatient department of the Babies' Hospital, the blood of 150 negro patients was examined and 13, or 8.66 per cent, gave latent sickling, while the blood from white children did not show this phenomenon. Cooley [10] found 7.5 per cent in Detroit, Graham and McCarthy [17] 7.2 per cent in Alabama, and Miyamoto and Korb [4] 6.3 per cent in St. Louis. As Sydenstricker [5] pointed out, sickle cell anemia causes a high mortality in young children, and according to Graham and McCarthy,[17] few of its victims survive the age of 50, the majority dying before 30.

The families of six of our patients were examined, and in five the blood of the mother showed latent sickling. In one family both father and mother had the trait. In case 3, two sisters, 5 and 6 years old, showed latent sickling in the blood, but were without symptoms. In another family a girl, aged 12 years, had sickle cell anemia, while her brother, aged 5 years, and her sister, aged 3 months, had the sickle cell trait. Another patient with active sickle cell anemia, aged 6 years, had a sister 7 years of age whose blood showed latent sickling.

---

12. Huck, J. G., and Bigalow, R. M.: Poikilocytes in Otherwise Normal Blood (Elliptical Human Erythrocytes), Bull. Johns Hopkins Hosp. **34**:390, 1923.

13. Lawrence, J. S.: Elliptical and Sickle Shaped Erythrocytes in the Circulating Blood of White Persons, J. Clin. Investigation **5**:31, 1927.

14. Castana, V.: I gigantocite e le anemie semilunari, Pediatria **33**:431, 1925.

15. Bernhardt, H.: Ovoläre Poikilozytose bei Begleitanämie, Deutsche med. Wchnschr. **53**:1161, 1927.

16. Emmel, V. E.: A Study of the Erythrocytes in a Case of Severe Anemia with Elongated and Sickle Shaped Red Corpuscles, Arch. Int. Med. **20**:586 (Oct.) 1917.

17. Graham, G. S., and McCarthy, S. H.: Sickle Cell Anemia, J. Lab. & Clin. Med. **12**:536, 1927.

LITERATURE

The literature on sickle cell anemia is almost entirely American, dating from Herrick's [18] description of the peculiarly shaped red cells in the blood of a colored man, 20 years old, who was observed in Chicago in 1910. Jaffe [2] has given a complete review through the year 1926. The first cases of sickle cell anemia occurring in children were reported by Sydenstricker, Mulherin and Houseal [11] in 1923. Both were negro boys, 5 and 6 years old. The autopsy on the younger child was reported in detail. His grandfather, father and brother showed latent sickling of the red cells, while in the family of the other boy both parents and two of his sisters showed the sickling. Later in the same year Huck [19] published his study from Baltimore, where he followed two families through three generations and found sickling in each generation. In 1926, Cooley and Lee [10] reported two cases in children, aged 3 and 5 years. They mentioned three autopsies on children whose blood showed sickling, but reported "no special findings either in the spleen, liver or bone marrow." Dryfoos [20] observed the case of a boy 3½ years old, and Anderson [21] a boy of 13 years, while Archibald [22] recorded the case of an Arab boy 10 years old whom he had observed in the Sudan. That the subject of sickle cell anemia is one of increasing interest all over the country is shown by nine papers published in 1927 in American journals, and one in a German journal by Jaffe,[2] who gave the most detailed and careful histologic picture of two patients who died of tuberculosis.

Since sickle cell anemia is a familial, hemolytic anemia, its differential diagnosis from familial hemolytic icterus is of interest. The chief point is the presence of small red cells in the one, and of sickle cells in the other. Fragility is usually, but not invariably, increased in hemolytic icterus and is normal in sickle cell anemia. Phagocytosis occurs chiefly in the liver in both, and iron pigment is present in both. The spleen is always large in hemolytic icterus, and in infants and young children with sickle cell anemia it may be large or normal. In older children or adults it may be small. The icterus is much deeper in hemolytic jaundice, and the icteric index much higher. Nucleated red cells are numerous in sickle cell anemia, and reticulocytes are numerous in both.

---

18. Herrick, J. B.: Peculiar Elongated and Sickle-Shaped Red Blood Corpuscles in a Case of Severe Anemia, Arch. Int. Med. **6**:517 (Nov.) 1910.

19. Huck, J. G.: Sickle Cell Anemia, Bull. Johns Hopkins Hosp. **34**:335, 1923.

20. Dryfoos, M.: Sickle Cell Anemia, Arch. Pediat. **43**:436, 1926.

21. Anderson, H. B.: Sickle Cell Anemia: Report of an Active Case, Am. J. M. Sc. **171**:641, 1926.

22. Archibald, R. G.: Case of Sickle Cell Anemia in the Sudan, Tr. Roy. Soc. Trop. Med. & Hyg. **19**:389, 1926.

Splenectomy had been suggested by Sydenstricker and by Huck,[19] but Hahn and Gillespie [7] were the first to report the result of the removal of the spleen in sickle cell anemia. The patient was 4 years old. In Stewart's [9] case, splenectomy was performed on a boy of 6 years. Bell, Kotte and Mitchell [3] reported splenectomy in a boy of 18 months, and Cooley and Lee [10] in a girl of 2 years 9 months. Finally, Hahn [8] reported a second case in a boy 18 months old. A cure did not result in any of these patients.

The term "drepanocytic anemia" has been suggested by Hahn,[8] while Miyamoto and Korb [4] would designate the latent phase as "meniscocytosis." Sickle-shaped red corpuscles was the descriptive term used by Herrick [18] in his original communication and sickle cell anemia was first used by Huck.[19]

SUMMARY

In New York, sickle cell anemia is as common as it is in cities of the South and West. In a series of fifteen negro children whose blood showed sickle cells, twelve were in the active phase and three were in the latest phase. In three of the active cases, the patients died of the anemia without other anatomic cause for death. All showed fatty degeneration of the myocardium and liver, distention of the sinuses of the spleen with sickle cells, phagocytosis of the sickle cells by Kupffer cells in the liver and iron pigment in the spleen, liver and kidneys. In two latent cases, the patients died of tuberculosis and in one the patient died of pneumococcus meningitis. Syphilis was present in one child of the series.

ALLERGY IN AN INFANT OF THREE WEEKS

REPORT OF A CASE AND COMMENT *

GEORGE M. LYON, M.D.
HUNTINGTON, W. VA.

A case of allergy in an infant, aged 3 weeks, is reported because of the rarity of the condition at that age and because of the interesting problems that are presented in the consideration of the condition.

REPORT OF CASE

History.—A. M. E., a white girl, aged 6 weeks, first examined by me on Feb. 8, 1926, had been suffering from an unusual form of swelling since her twenty-first day.

The family history is of interest only in that an entirely similar condition had not been known in the family of either parent. One maternal uncle had had severe eczema from early infancy. A second maternal uncle had suffered from asthma. There was no history of syphilis in the family.

The patient was delivered normally at full term and had been given nothing but breast milk, the mother having had an abundant supply. The infant gained weight steadily, and nothing remarkable was noticed until the twenty-first day. At this time the entire left leg and the left labial folds were tensely swollen to approximately twice the size of the corresponding parts on the right. The swelling, as described by the parents, was red and tense. The patient was only slightly fretful and nursed normally. This swelling persisted for five days, and, as it subsided, the left side of the face, the right arm and the right pectoral and right scapular regions became swollen, red and tense with a similar swelling. The swelling of the left leg and labial folds had disappeared; otherwise, the general condition remained unchanged. Then for the first time a physician was consulted, and a diagnosis of erysipelas was made and local treatment given accordingly. After three days the second area of swelling disappeared, only to be followed by a similar swelling of the right leg and both labial folds and the right buttock. During the three weeks previous to admission, this condition existed, the swelling disappearing from one place, only to appear at another, about every third or fifth day.

Examination.—When the infant was seen, she was nursing vigorously, was apparently happy and was well nourished. The stools as seen during this examination were normal for a breast-fed infant. The temperature by rectum was 98.4 F. and during the entire period of observation did not exceed 99.2. The entire left leg and hip, the left buttock and both labia were tremendously swollen and red, and were little if any warmer to the touch than the surrounding normal areas. On pressure there was little pitting. A clearcut line of demarcation was present at the margin of the swollen areas. When the fingers were passed from the normal to the involved areas, there was the same sensation of marginal elevation and turgidity as is found in erysipelas. The labia were so swollen that it was impossible to get the thighs together. Swelling of the right side of the face and of the right arm and shoulder was present, which the parents said had been

* Received for publication, June 8, 1928.

* From the Department of Child Hygiene, Marshall College.

there for four days. This, they said, had greatly receded but was easily discernible on examination. Other significant physical symptoms were not present.

The laboratory studies revealed little. The blood of both parents and of the patient gave a negative Wassermann reaction. The leukocyte count of the patient was 8,500, with 54 per cent lymphocytes and a polymorphonuclear eosinophilia of 6 per cent. Abnormal cell forms were not found. The roentgenograms of the long bones did not show evidences of syphilis or scurvy, or other abnormal conditions.

Clinical Course.—The next day, February 9, the swelling was entirely gone from the right side of the face, the right shoulder and the right arm. It was unchanged on the left side, being still quite tense and red but not hot to the touch.

The third day, February 10, the condition was the same as on the second day, and the following interesting facts were elicited: The mother's diet was poorly balanced. Although she lived on a farm and kept a cow, she drank little milk and ate little if any butter or eggs. She had not eaten any green vegetables since the infant's birth, nor had she had any beefsteak, liver or red meats. Pork and bacon, occasionally supplemented with chicken, were the only forms of meat she had had all winter. As is frequently the case in the inaccessible, rural mountainous regions, corn bread and dried white navy beans had been the main source of food. On many days, she had eaten these three times. This diet was supplemented at times with potatoes, onions, canned corn and, rarely, some cereals. As the infant was receiving only breast milk, it was suspected of being responsible for the allergic phenomena observed. With the impression that the condition was an angioneurotic edema, possibly caused by the breast milk or some factor of the breast milk, the protein skin tests were made with the following results: The infant was extremely sensitive to skin tests of its own mother's milk, slightly sensitive to that of another mother and did not react to the dried breast milk of two other mothers. The infant reacted positively to dried white navy beans and dried sweet corn, and the Arlington Chemical Company's navy bean and corn proteins. She did not react to cow's milk, casein, hen's egg (whole), beef, potato, wheat or oats. Similar protein tests performed on the skin of the mother showed uniformly negative results.

In view of the foregoing tests, an attempt was made to eliminate beans and corn from the mother's diet, and the infant was continued on the breast. The mother was given a liberal diet, including cow's milk, eggs, green vegetables, oranges and cod liver oil. She was instructed to have beefsteak, beef kidneys and sweetbreads whenever possible.

In an attempt to clear up the local condition, as well as to study the effect of epinephrine on it, 5 minims (0.3 cc.) of epinephrine solution (1 : 1,000) was given hypodermically. Shortly after, an appreciable change occurred in the discoloration of the swollen areas. In one hour this dosage was repeated, and thereafter epinephrine chloride solution of the same strength was given by mouth every four hours. In less than thirty-six hours, on the night of February 11, the swelling was entirely gone, and new regions were not involved. Four days later the epinephrine by mouth was withheld purposely, and within twelve hours the left side of the face and the left shoulder and the labia were again beginning to swell and get red. The epinephrine was withheld for thirty-six hours. During this time the swelling and redness progressed, and on February 16 the epinephrine solution (1 : 1,000), 10 minims (0.6 cc.) by mouth every four hours, was again given and within twenty-four hours the improvement was remarkable.

The infant was discharged from the hospital on February 20, without any evidence of swelling or discoloration. Thereafter, the epinephrine was withheld once purposely at my direction and a second time, when the supply ran out and the parents were unable to get over the twenty miles of bad mud roads to replenish it. Both times there was a return of the swelling and discoloration within twenty-four hours. Gradually it became possible to increase the interval between doses of epinephrine, until four months later, at the age of 5½ months, the infant was able to go without it. Meanwhile, the mother was supposed to be getting a better balanced diet which was free from corn and beans. She was taking milk, butter and eggs more frequently.

It was difficult to understand why it was necessary to continue the epinephrine solution for so long a time. Although the mother had insisted for two years that she had adhered strictly to my diet prohibiting beans and corn, at the suggestion of Dr. W. W. Duke, I again inquired into this phase of the case more stubbornly, and the mother finally admitted the following facts: Until the infant was 5½ months old (June, 1926) the mother had been forced by necessity to take beans and corn at times (two or three times a week instead of two or three times daily). During this period, the swelling or "hives" occurred each time after the mother ate beans or corn. The epinephrine solution by mouth for twenty-four or thirty-six hours and the avoidance of beans or corn for a few days was always followed by a clearing up of the swelling. When the infant was 5½ months old, the mother was able to cease entirely the taking of beans and corn, as green vegetables were available from her own garden. The swellings did not appear on the infant any more, although she was not weaned until two and a half months later, at the age of 8 months. She continued to do well, and swelling or "hives" did not appear after she was 5½ months old, the time when the mother really stopped eating corn and beans.

At the suggestion of Dr. Duke, the infant was given white navy beans, when she was a little over 2 years of age. Within four hours, giant urticarial lesions appeared all over the body. The day this test was made, cow's milk and white navy beans were the only articles of food allowed. Even butter and salt were not given. The results seemed conclusive as to the continued sensitivity to white navy beans. Skin tests were again positive to white navy beans and corn, although not so violently positive as two years previously. The infant had received fresh string beans on numerous occasions, without allergic manifestations.

A further study was then made to ascertain whether the child was sensitive to the milk from a cow fed liberally on meal made from white navy beans, canned corn and corn meal. The cow was kept on this diet for two weeks. During the second week, the patient's sole source of milk was from this cow. The milk was taken fresh and unboiled. From 24 to 32 ounces (710 to 946 cc) of milk was taken daily, and yet allergic manifestations did not occur. A few days after the completion of this experiment, she was again given mashed white navy beans and the urticaria appeared as usual after such ingestion.

COMMENT

Talbot [1] reported a case in which severe eczema in a nursing infant cleared up when the mother discontinued eating chocolate, and recurred when she ate it again. In nursing infants with eczema, O'Keefe [2]

1. Talbot, F. B.: M. Clin. N. Amer. **1**:985 (Jan.) 1918.
2. O'Keefe, E. S.: Boston M. & S. J. **182**:569 (Nov.) 1920.

observed positive skin reactions to foods which they had never eaten. Shannon [3] found that withdrawing from a nursing mother's diet certain foods to which she was immune, but to which the infant was sensitive, was followed by the disappearance of an urticarial eruption on her nursing infant. With anaphylactic experiments in guinea-pigs, he demonstrated the secretion of allergens in human breast milk.

Kolmer [4] stated that sensitization to foods is acquired, although a predisposition to sensitization may be inherited. If active sensitization is not inheritable, then it must be assumed that infants become sensitized by food substances in the mother's milk in order to explain specific sensitization to foods which the child has never eaten. In the cases reported by O'Keefe,[5] not one of the mothers was sensitive to the protein to which her nursing infant was susceptible. Shannon [3] reported a similar experience in this respect.

In speaking of eczema in the exclusively breast-fed infants, O'Keefe [6] said:

> More than 40 per cent showed a positive reaction to egg proteins, 39 per cent to cow's milk, 5 per cent to oat, and about 2 per cent to wheat. Fourteen per cent responded to both egg and milk protein. I found no patient showing sensitization to human milk proteins, nor did I find any mother showing a skin reaction to the protein to which her child responded. The obvious conclusion seems to be that the breast milk in these cases contains a foreign protein which the child has ingested and absorbed unbroken, and sensitization has finally resulted. Of this sensitization the eczema is a manifestation.

It was interesting to note that in the case here reported, the infant was sensitive to the breast milk of her own mother, slightly sensitive to that of another mother and gave no reaction to the breast milk of two other mothers. May there not be a great difference in the ease with which certain mothers may secrete these allergens? May this not be the reason that the allergens appear in the breast milk and produce an early sensitization in the nursing infant, later to be followed by allergic phenomena?

In this instance at least, there has been clinical evidence of epinephrine being absorbed from the mouth, pharynx or upper gastrointestinal tract. The generally accepted view is that epinephrine is

---

3. Shannon, W. R.: Demonstration of Food Proteins in Human Breast Milk by Anaphylactic Experiments on Guinea-Pigs: Their Probable Relationship to Certain Diseases of the Nursing Infant; A Preliminary Report, Am. J. Dis. Child. **22**:223 (Sept.) 1921.

4. Kolmer, J. A.: Infection, Immunity and Biologic Therapy, Philadelphia, W. B. Saunders Company, 1924, p. 668.

5. Footnote 2. O'Keefe, E. S.: A Dietary Consideration of Eczema in Younger Children, J. A. M. A. **78**:483 (Feb.) 1922.

6. O'Keefe (footnote 5, second reference).

ineffective when administered orally.[7] In infants, small doses of from 5 to 10 minims may have a beneficial effect when placed carefully under the tongue, although it is doubtful if the effect is nearly as constant as by hypodermic effect. The effect is not so transitory when obtained. Foote [8] referred to the use of oral administration of epinephrine as being effective in bronchial asthma at times. In cases of early allergy such as one may see in early infancy, epinephrine by mouth may be a valuable adjunct in the therapeutic field. Many times it is impossible to remove or even locate the particularly active allergen. In such cases, the oral administration of epinephrine seems distinctly to be indicated.

SUMMARY

The interesting features of this case may be summarized as follows:

1. The observation of the secretion of allergens in mother's breast milk with resulting allergic manifestations in an infant sensitive to those allergens.

2. The sensitivity in an infant and the lack of sensitivity in the mother as evidenced by the allergic reactions and protein skin tests. This is practically the rule and probably explains the occurrence of early acquired, so-called "inherited sensitivity."

3. The influence of the epinephrine solution (1:1,000) on the condition when administered orally.

4. The sensitivity to white navy beans present in the child after two years, showing there has not been complete desensitization.

5. The inability to demonstrate the secretion of these allergens in the milk of a cow fed liberally on a diet containing white navy beans and corn.

6. A sequella to an incomplete diet, which has not previously been pointed out. The diet was incomplete mainly because of the poor geographic and poor economic surroundings of the family.

7. The fact that the child reacts to white navy beans but can eat fresh string beans without allergic manifestations suggests that there is a different protein structure or "body" in the two foods.

8. The use of epinephrine solution with the withdrawal of the offending proteins from the mother's diet gave the best results.

9. This case is reported as being one of early acquired rather than inherited or congenital sensitivity.

1015 First Huntington National Bank Building.

---

7. Wearn, J. T.: Pharmacology of Epinephrine, J. A. M. A. **83**:1508 (Nov. 8) 1924.

8. Foote, J. A.: Abt's Pediatrics, Philadelphia, W. B. Saunders Company, 1924, vol. 3, p. 922.

INTUSSUSCEPTION DUE TO PAPILLOMA IN A CHILD OF TWENTY-ONE MONTHS*

EDWARD J. LAMB, M.D.
SANTA BARBARA, CALIF.

Papillomas of the rectum are not of infrequent occurrence, and many cases have been reported. Only a few cases of papillomas of the colon have been reported. Papillomas of the small intestine, however, are rare. There is little to be found in the literature of the last twenty-five years on this condition.

Ward [1] reported a case of recurrent enteric intussusception in a child due to a simple tumor (adenopapilloma). This is the only case of intussusception caused by a single tumor that I could find reported after an exhaustive search in the literature of the last twenty-five years.

Because this condition is so uncommon and obscure, the following case is reported:

REPORT OF CASE

History.—Shirley B., aged 21 months, white, born Sept. 1, 1924, in the United States, of American parents, was referred to me on June 7, 1926, by Dr. A. Isaacs of Santa Barbara. The family history was negative. The parents were in good health. There was one brother, aged 7½ months, who was in good health. There was no history of miscarriages or familial chronic diseases.

The infant was born two months prematurely by normal delivery. Her neonatal and infantile health was good. She had been breast fed for six months and later fed a formula of milk, water and Dextri-Maltose. She walked at the age of 14 months, and cut teeth at the age of seven months. She had not had any contagious disease, nor prophylactic treatment and the only sickness had been frequent attacks of bronchitis. The infant did not talk at the time of examination.

"Teething" and vomiting were the chief complaints. For the past three or four months, the infant had had "colic." During the last three weeks she had not been as well as usual, but the movements of the bowels had been normal. She began to vomit about one week before presentation at the clinic. At this time she became constipated; enemas, mineral oil and fruit juices were given without any effect. Three or

* Received for publication, June 20, 1928.
* From the Children's Clinic, Santa Barbara, Calif.
* Read before the Southwest Pediatric Society, May 9, 1928.

1. Barrington-Ward, L. E.: Brit. J. Surg. **12**:800 (April) 1925.

four days before admission to the hospital, the abdominal pains became more constant and were accompanied by marked flatulence.

Examination.—Examination revealed a white female child, 21 months of age, poorly developed and poorly nourished, weighing 24½ pounds (10.9 Kg.). She was 32 inches (81.2 cm.) in height and had a good disposition; she was not fretful, but rather stoical. The facies expressed transient pain at two to three minute intervals.

The results of a general examination were negative, except that the abdomen was moderately distended and markedly tender, especially in the right upper quadrant. Spasm and rigidity were marked at this quadrant and a palpable sausage shaped mass was also found. During this tactile examination of the abdomen, the baby gave no evidence of severe pain, or crying or fretting, but at two to three minute intervals she strained and winced as if in acute pain. The liver and spleen were not palpable. There were no umbilical or inguinal hernias. The genitals were normal. The temperature was 100 F. and the pulse rate 130.

Examination of the blood revealed white blood corpuscles, 18,500; polymorphonuclear leukocytes, 74 per cent; lymphocytes, 26 per cent. Urinalysis showed a trace of diacetic acid and of indican, and a large amount of acetone. Blood was not seen in the stools at any time until streaks of bright red blood were found during an enema given at the Cottage Hospital one-half hour before operation.

Operation.—A diagnosis of intussusception was made, and the patient was referred to Dr. R. Brown with recommendation for immediate operation. Operation was performed within three hours after the infant was first seen, and revealed the following condition:

The abdomen was filled with free fluid, and the appendix was normal. An intussusception of the small intestine was found about 1 foot from the iliocecal junction. The telescoped section was restored to normal continuity. In the intussusception was found a tumor mass the size of a large olive. On the mesenteric border of the mass was an indented area suggestive of old scar tissue. The section of bowel containing the tumor mass was resected and the ends distal and proximal were closed. A side-to-side anastomosis was then performed on the loops of the bowel immediately adjacent to the resection. The wound was closed without drainage.

Pathologic Report.—The pathologic report on the specimen was made by Dr. Frank Nuzum, pathologist. The tissue was found to be that of a polypoid tumor from the small bowel. On section, this tumor was found to consist of a dense stroma of connective tissue cells with small nuclei and with a mucinous appearance. Scattered through this substance were numerous blood channels and diffusely spread red blood

cells, lymphocytes and polymorphonuclears. There were also to be seen distinct endothelial cells with large nuclei and a considerable amount of cytoplasm. There was no evidence of excessive activity of endothelial cells or of other evidences warranting diagnosis of a malignant condition. The condition was characteristic of, and the diagnosis was, benign intestinal polyp.

NOTE.—This is a rather unusual condition and frequently multiple polyps are present.

Progress.—The child made an uneventful recovery and was dismissed from the hospital on July 4, 1926, with the wound healed, the appetite good and the movement of the bowels regular. Her weight on dismissal was 25¼ pounds (11.3 Kg.). She made a progressive gain in health; the hemoglobin of the blood was increased and her weight on Feb. 7, 1927, eight months after her operation, was 33 pounds (15.0 Kg.), or a gain of approximately 1 pound (0.5 Kg.) a month. She has not had any attacks of gastric or abdominal distress, and the movement of her bowels is kept regular by a diet rich in fruit, also mineral oil. A letter received on April 28, 1928, almost two years after operation, stated that Shirley had been wonderfully well, was full of life and weighed 37 pounds (16.8 Kg.), a gain of over 12 pounds (5.4 Kg.) in less than two years.

COMMENT

Papillomas of the small intestine are rare and intussusception caused by papilloma is of rare occurrence. When polypoid growths occur in the intestine, they are apt to be multiple. Recovery of an infant after resection for invagination ileus is a surgical curiosity.[2]

2. Peterson, E. W.: Surg. Gynec. Obst. **35**:436 (Oct.) 1922.

Progress in Pediatrics

SINUSITIS IN CHILDREN*

SIMON L. RUSKIN, M.D.
NEW YORK

The various textbooks on pediatrics give the impression that sinusitis in children is relatively rare. The anatomic fact that at birth there are already two well formed sinuses, the antrum and the ethmoid, together with the frequency of nasal discharge in children, leads one to suspect that perhaps sinusitis is present among children much frequently than is now diagnosed.

The law postulated by Politzer for the middle ear is probably equally true for the nose. Politzer believed that in every instance acute otitis simultaneously involved the accessory air spaces in the mastoid to a greater or lesser degree. I believe that in every instance acute rhinitis involves the accessory nasal sinuses in a similar manner. The anatomic conditions in the nose are somewhat better for drainage in the nasal accessory sinuses than in the ear and so proportionately less severe cases are seen. The frequency of sinusitis must be greater than that of otitis media.

Even at birth, one finds evidence of the presence of a recess extending into the maxilla, forming a small maxillary sinus. In addition, one also finds a number of small, distinct ethmoidal cells. These rudimentary sinuses become quite appreciable during the first and second years. The remaining space of the maxilla is filled by the germinal teeth and spongy bone. Even after the eruption of the milk teeth, but little space remains for the maxillary sinus. With the beginning of the second dentition, that is about the seventh year, the maxillary sinus grows rapidly. It attains a fair size after the eruption of the molars and the increase in height of the skull.

The frontal sinuses, according to Stiner, develop during the first or second year, and at the sixth or seventh year have attained the size of a pea. They attain a definite size usually between the fifteenth and twentieth year.

The sphenoid sinus, which Toldt really considers a posterior ethmoidal cell, usually develops about the seventh year. How frequently these small sinuses can be affected is gathered from Hark's statistics. In 394 autopsies of children in which he had opened the accessory

* Received for publication, July 18, 1928.

* Read before the Section in Pediatrics, New York Academy of Medicine, February, 1927.

nasal sinuses, he found evidence of disease in sixty-two cases, in fifty-two of which the children ranged between 9 months and 15 years of age. In this group, the maxillary sinus was affected in forty-seven instances; the ethmoids in three, and the sphenoid twice. The percentage of cases of sinusitis in children who do not succumb to fatal diseases, must, of course, be much greater. In children brought for examination on account of a chronic cough, a continuous irregular temperature, without any apparent reason, a cervical adenitis or recurring attacks of otitis media, it is surprising how often x-ray examination of the sinuses will show a distinctly cloudy antrum or ethmoid.

In Onodi's [1] tabulation of cases of severe disease of the accessory nasal sinuses reported by various investigators, he found scarlet fever to be the most frequent cause. Onodi also observed that in the disease of the ethmoid cells there was usually associated involvement of the frontal and maxillary sinuses.

In considering the pathologic conditions of these sinuses, one must study three avenues of invasion from the sinuses: (1) the possibility of the absorption of toxic products; (2) the direct infection of the blood stream from the nasal sinus, and (3) infection of the cranial lymphatics. Each produces a definite clinical picture.

In the type of case caused by the absorption of toxic products, there is a history of repeated colds, accompanied by purulent nasal discharge, often with a history of repeated bronchitis, recurrent pneumonia or persistent cough, associated frequently with gastric disturbances. The patient, although appearing well nourished and frequently of good color, has periods of paleness associated with puffiness of the eyelids and sleepiness, but he is, nevertheless, often restless at night. This condition will last a few days, and then clear up promptly, only to recur a few weeks later, going through the same cycle. This variety of case in more advanced conditions gives a typical picture of parenchymatous nephritis so excellently described by Clausen and Marriott. Marriott [2] says, "In practically all of the cases of severe parenchymatous nephritis which we have observed, there has been a staphylococcus infection of the nasal accessory sinus, and especially of the maxillary sinus. We have regularly observed that increased activity of the infection was accompanied by an exacerbation of all of the symptoms of nephritis and further successful treatment of the nasal sinus infections was regularly followed by rapid improvement and by complete disappearance of all the symptoms of nephritis. Forms of treatment other than those directed toward the infection were of but temporary benefit. The causal relation between infection and nephritis in this case seems definite." These

1. Onodi, A.: Die Nebenhöhlen der Nase Beim Kinde, 1911.

2. Marriott, McKim: The Nature of Nephritis, Northwest Med. **24**:472 (Oct.) 1925.

patients show a slight fluctuation in temperature, ranging at times from 99 to 100 and even 101 F., and in this case the temperature may continuously stay elevated until the nasal infection is successfully combated.

This picture is much different from the second avenue of invasion, the type of sinusitis which is apparently associated more with an infection of the blood stream than with a toxic absorption. The children become acutely sick, with nasal obstruction and discharge; the temperature rises to 104 and 105 F.; the child is prostrated, and the nose, throat and pharynx seem simultaneously involved. The mucous membrane of the nose and pharynx has a beefy redness and is moderately thickened, especially behind the posterior pillars of the tonsils. There is a slight tendency to edema; the patients have a considerable pallor, and they occasionally show evidence of acute glomerular nephritic changes, according to Marriott, apparently the result of the action of the streptococcus toxins. In these cases the capillaries throughout the whole body are affected, especially in the gastro-intestinal tract. I have seen this type of case occurring at times in almost epidemic form. The pus characteristic of empyema of the sinus is not seen in this type, but is rather a tenacious mucopus filling the postnasal space and hanging on the pharyngeal wall. The patients at times appear septic, and have an irregular temperature for a period ranging from a few days to several weeks or more. Usually they recover completely, no signs remaining of empyema of the sinuses. This is the variety of sinusitis that is the source of most of the acute infections of the ears, and explains the frequency of the presence of *Streptococcus hemolyticus* in acute conditions of the mastoid in children. The beefy swelling of the mucosa of the nose and throat extending into the eustachian tubes occludes them, thus sealing the products of inflammation in the tube and middle ear and producing a vicious cycle that goes on to the involvement of all the accessory air spaces in the mastoid. The value of early paracentesis in these cases becomes apparent. Waiting for spontaneous rupture is equivalent to waiting for more extensive involvement. The ear under these conditions is equivalent to a paranasal sinus with an extensive labyrinth of cells having one drainage opening, the eustachian tube, placed between the infected sinuses and adenoids. The status of the ear under these circumstances is much less favorable than that of the paranasal sinuses and one finds, accordingly, that surgical intervention is so much more frequently imperative for the ear than for the paranasal sinuses.

The third variety of disturbance seen originating from the lateral nasal wall and paranasal sinuses is inflammation of the cranial lymphatic glands. Without going into a consideration of the entire mechanism of lymphatic drainage of the nose, I want to bring out the point that the

retropharyngeal lymph nodes in children drain the lateral nasal wall, accessory sinuses and eustachian tube. These nodes are found in front of the deep cervical muscles. Two groups are found. The most important is the lateral group, arranged in typical, symmetrical distribution. Usually, there are one or two such nodes on each side, or there may be three or more on each side, arranged in the form of a chain. These retropharyngeal glands are always present in children, but frequently absent on one side in adults. Their typical location is medial to the internal carotid near its entrance into the cranium. Anteriorly, they lie close to the soft palate near the palatopharyngeal arch. During inflammation of these glands, the pharyngeal wall swells close to the faucial tonsil, assuming the appearance of a round tumor. The location is fairly constant. In small children, variations of small intermediate glands occur in the retropharyngeal space. These may be more medial or even in the midline.

In the cases in which one is dealing with sinusitis of the staphylococcus variety, one usually finds slight enlargement of the glands, but in the second type of case of acute sinusitis of the form usually diagnosed as nasopharyngitis, with *Streptococcus hemolyticus* infection one sees at times a severe lymphadenitis, with the typical pharyngeal swelling, simulating retropharyngeal abscess.

The history of this type of case will reveal that the patient suffered from an acute cold for a few days. If the patient was not treated, or was exposed to undue chilling, an exacerbation in the cold occurred, followed by a thick, pale greenish mucoid discharge, high temperature, loss of appetite and subsequent swelling of the retropharyngeal lymph nodes, with difficulty in swallowing. Some of the cases will go on to suppuration; most of them, however, will resolve under appropriate treatment. In some of these cases, there may be more or less permanent enlargement of the retropharyngeal glands. I wish to repeat that in these cases one is dealing with an acute sinusitis with secondary and almost simultaneous lymph adenitis with involvement of the epipharynx, pharynx and tonsils. Diseased tonsils and adenoids are probably a frequent source of the infection. In cases in which there has been a thorough removal of tonsils and adenoids, and in which there is a history of repeated nasal colds, I suggest that a more accurate diagnosis be made in those cases that have been classified under the heading of "nasopharyngitis."

An extremely interesting phase of the subject of sinusitis in children is what I call "post-tonsillectomy sinusitis." The mother will tell you that the child, prior to tonsillectomy, may have suffered from repeated sore throat and mouth breathing, but that following the removal of the tonsils and adenoids there has not been any improvement in breathing

—in fact, that the child suffers more from nasal discharge than ever before. Although there may not have been any cough previous to the operation, the child may develop a chronic cough, especially at night, or a mild or severe asthmatic attack. Instead of improving in weight and appearance, the child now looks distinctly pasty, with a tendency to the formation of slight edema of the lower lids which the parent will describe as "rings" under the eyes. In short, the patient does not appear to have been at all benefited, but rather to have lost ground following the operation. Examination of the throat will usually reveal that an exceptionally clean tonsillectomy was performed without any evidence of tonsil or adenoid rests having been left over. The patients will give a history of having had several operations on the adenoids without any improvement. In fact, I have met pediatricians who, having been impressed by such cases, will argue against tonsillectomy altogether. The analysis of this condition is indeed interesting.

The anatomic relationship of the adenoid is the key to the entire situation. A child suffering from hypertrophied tonsils and adenoids frequently has associated with these conditions a moderate or fairly severe grade of sinusitis. In examining anatomic material from children one is struck by the relationship existing between the size of the adenoid and the epipharynx. In a large percentage of cases, the hypertrophied adenoid occupies practically the entire postnasal space. The children are, of course, mouth breathers, and the textbook picture of their facies is classic. The nose in these cases is practically separated from the pharynx. The discharge from the sinuses will, as a rule, stay in the nose, there being little air current in either direction. The adenoid here acts effectively as a postnasal tampon. What happens when the adenoid is removed? The nasal discharge drains easily during the night into the pharynx, and some of the discharge is swallowed during sleep while some drains into the larynx and trachea. The child develops a cough in clearing his larynx and trachea and an upset stomach from the pus swallowed. His general resistance is impaired, and he suffers more from repeated colds than ever before. He may become sensitive to the bacterial proteins or swallowed discharges and develop a true asthma. The patient is now caught in a vicious cycle. Here one must intervene actively by combating the sinusitis. An x-ray film will usually reveal a chronic maxillary sinusitis, a chronic ethmoiditis or both. Occasionally, in older children, it may be the sphenoid sinus. Appropriate treatment in these cases will give the most gratifying results.

The trouble arises partly from the point of view of the pediatrician or practitioner who refers the child for tonsillectomy and adenoidectomy as a cure for the upper respiratory condition. In many cases, the

operation must be considered as a preliminary step to establishing normal physiologic conditions in the nose. I, therefore, make it a practice to examine within one month, all children who have been operated on, in order to determine whether the sinuses need further attention.

The question of treatment in these children is difficult and trying on the patience of both the physician and the family. Nevertheless, in order to get satisfactory results in the treatment of paranasal disease in children, it is frequently essential that energetic measures be used. Conservatism must, however, be the keynote. It must be borne in mind that these little patients usually have a much lowered resistance and are poor subjects for operative intervention. They are also extremely susceptible to intercurrent infections. Any patients requiring surgical intervention should be isolated more for their own protection than for any danger that they may cause their neighbors. The first requisite in these cases is the exact diagnosis. One must determine whether one is dealing with a chronic sinusitis, with an acute sinusitis, or with an acute exacerbation of a chronic sinusitis.

The local treatment is dependent on the diagnosis of the local condition. The best aid in the diagnosis is the x-ray. In very young children intranasal examination is more or less unsatisfactory. In older children, however, one can get a fairly good idea as to whether one is dealing with the anterior series of sinuses, or the posterior series of sinuses. When once one encounters a true case of sinusitis, for instance, in the maxillary, sphenoid, ethmoid or frontal sinus, the time-honored custom of instilling drops in the nose is of little value. I have yet to see a chronic case in which the patient was decidedly benefited by the instillation of drops into the nose. When there is an empyema of the antrum, the direct puncture and irrigation yields brilliant and rapid results. Some of the children whom I have demonstrated this evening have had months of treatment with intranasal drop instillations with practically no nasal improvement. Instead, they have suffered frequent gastro-intestinal disturbances to some extent, from the continuous swallowing of the medicaments. It is amazing to see how promptly improvement follows the washing out of a chronically infected antrum. The procedure of the antrum puncture is simple, if one is thoroughly acquainted with the regional anatomy and uses the correct instruments. In fact, the puncture of the maxillary sinus in children is almost identical with that in adults. The procedure takes but a few minutes and can be comfortably performed under ethyl chloride, or light ether anesthesia. For irrigation I use 1 to 5 solution of hexyl resorcinol S T 37. X-ray reports show empyema of the maxillary sinus in about 75 per cent of the cases of chronic sinusitis. The other sinuses are more difficult to approach for treatment and less frequently involved.

In cases of ethmoid or sphenoid sinusitis, nasal irrigation is valuable. About 1 pint of the foregoing solution is used, the inflow tube being in one nostril and the fluid circulation through the nasal fossae and leaving through the opposite nostril. Care must be taken not to occlude either nostril. Instead of a nasal tip, I use a small glass tube for the inflow. In older children, the irrigation may be followed by a light bath treatment such as described by Brunnings in the treatment of sinusitis in adults. The heat in this apparatus shrinks the mucosa and permits much better drainage without the subsequent engorgement that is usual in shrinkage from epinephrine hydrochloride. Some of these children also respond to the milder procedure of nasal packing with mild silver protein, followed by a thorough nasal irrigation such as I have just described, or the use of simple suction with either a hand bulb or a suction apparatus. The suction should not exceed 10 inches (4.5 cm.).

The treatment of the acute sinusitis is essentially conservative, and operative intervention is warranted only in the presence of threatened complications. Here one is dealing with acute rhinitis and sinusitis, associated in many cases with lymphadenitis. The mucosa of the nose and throat is thickened and engorged, leaving at times little more than capillary spaces between the turbinals and septum. The current method of instilling various oil drops or inert liquid petrolatum is sufficient completely to occlude the nose and stop what little ventilation was present. These oil drops may be useful in the chronic cases of sinusitis, in which the secretion has dried in clumps at the anterior nares, and the softening of the crusts by the oil allows them to be extruded during sneezing, or washed out during weeping; but in the acute cases they are more of a hindrance than a benefit. They are not sufficiently antiseptic to be of value, nor do they physiologically produce better ventilation. It is a common occurrence to see the little patients gasp for air and breath through the mouth after the instillation of the drops. The benefit of inhalation is much more definite, especially with the addition of a menthol-carrying substance. I find inhalations of 1 drachm of chlorbutanol (chloretone inhalant) to 1 pint of steaming water useful. An ordinary small electric vaporizer placed alongside the bed works excellently. The parent must, however, be warned to watch the apparatus. The chlorbutanol is more effective than either tincture of benzoin or simple steam. Inhalations of menthol should not be used in children under 2 years of age, because of the tendency to induce laryngeal spasm. In these cases atropine is useful.

SUMMARY

1. Anatomically one must consider disease of the paranasal sinuses a condition affecting children at all ages.

2. Sinusitis in children produces three distinct clinical pictures: one caused by toxic absorption in chronic sinusitis; a second, by infection of the bloodstream in acute sinusitis, and a third, by infection of the cervical lymphatics.

3. Treatment in chronic sinusitis must be directed exactly to the sinus involved, and since empyema of the maxillary sinus is most frequent, irrigation of the antrums is the most useful measure.

Fig. 1.—Left maxillary sinusitis.

4. Treatment in acute sinusitis should be conservative, with inhalation as the most useful procedure.

5. Prophylaxis is possible through the proper diet of the child, a normal calcium metabolism being maintained, and the careful avoidance of capillary shock through chilling.

REPORT OF CASES

CASE 1.—*Recurrent bronchitis and bronchial asthma relieved by puncture of the antrums and irrigation.*

M. G., aged 8, came to me three and one-half years ago with the history that shortly after the removal of his tonsils and adenoids, at the age of 2½, he

began suffering with nasal discharge, slight cough and loss of appetite. He had been operated on again for the removal of the adenoids but none were found.

Examination of the boy, at the age of 4½, when he first came to me, revealed pus in both nasal fossae; pus was also visible on the posterior pharyngeal wall. The tonsils and adenoids had apparently been completely removed. The child, although of good weight, appeared pasty, with a distinct puffiness which gave a ringlike appearance to his lower lids.

Fig. 2.—Bilateral maxillary sinusitis.

I proceeded to give him local treatment consisting of mild silver protein packs, and nasal suction, with but moderate relief of the symptoms. X-ray pictures of the sinuses showed a bilateral maxillary sinusitis. I recommended puncture and irrigation of both sinuses which the mother refused to allow because of the objection of her pediatrician.

On Jan. 29, 1927, the patient again appeared at my office. This time, in addition to his former symptoms, he suffered from intermittent attacks of vomiting, restlessness and severe attacks of bronchial asthma. The asthmatic attacks had become so frequent as to give the child little rest during the day or night.

I then proceeded to puncture and irrigate both antrums under light ether anesthesia, using a 1 to 1,000 solution of mercurochrome-220 soluble for the

irrigation. From both antrums, a thick, viscid mucopus was evacuated. The asthmatic attacks ceased within a week after the irrigation. The boy's general condition improved splendidly. The vomiting and restlessness disappeared. In February, 1927, at the time of the reading of this paper at the New York Academy of Medicine, I demonstrated him to the pediatric section of the society. At that time, he already showed good color and a marked diminution in the puffiness of his lower lids. To date, he has not had any recurrence of the asthma or sinusitis.

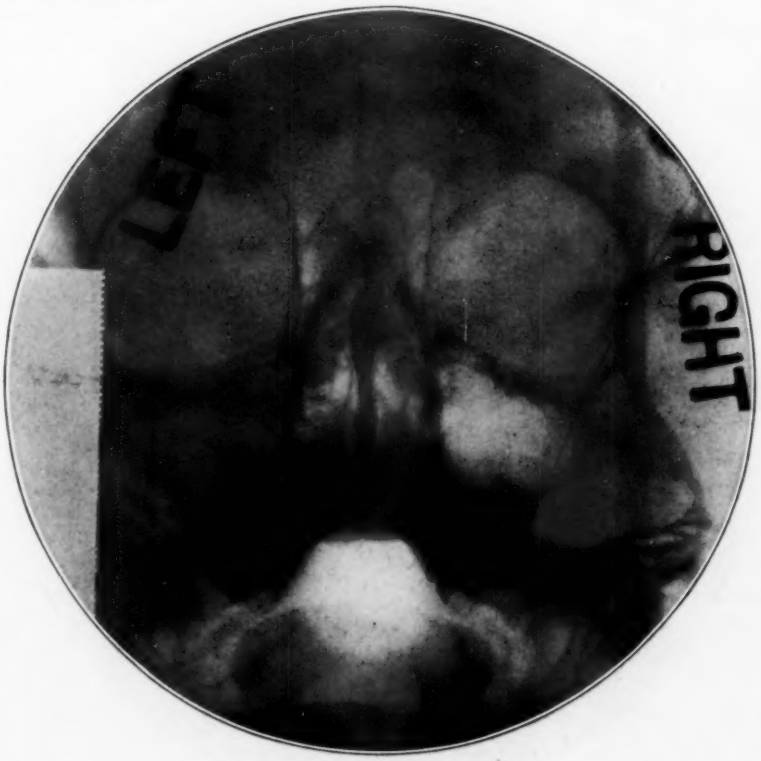

Fig. 3.—Left maxillary sinusitis.

CASE 2.—*Inability to gain weight, puffiness of the eyelids.*

N. D., aged 5½ years, came to my office on Oct. 7, 1926, because of poor appetite and inability to gain weight. There was not any cough or vomiting, and the bowel movements were regular. The child had a distinctly pasty complexion, with slight edema of both lower lids.

Examination of the nose and throat revealed pus in both nasal fossae, and diseased and hypertrophied tonsils. The following week, the tonsils and adenoids were removed. About two months later, the child again appeared for examination. At this time, he did not show any gain in weight, and in addition had a slight cough. An x-ray picture of the sinuses revealed a clouding of both antrums. Both maxillary sinuses were then punctured and irrigated with a 1 to 1000 solution of mercurochrome. Ten days later, the child showed marked

signs of improvement, the cough had disappeared and the appetite was decidedly improved. Six months later, the child was robust looking and gaining weight continuously.

CASE 3.—*Irregular temperature and headaches.*

R. M., aged 7, came to me on Jan. 4, 1927, because of frequent irregular rises in temperature lasting for several days and recurring without any apparent reason. He complained also of occasional sudden headaches, associated with loss of appetite and occasional vomiting. Both lower eyelids were distinctly puffy, and his complexion was sallow.

Fig. 4.—Bilateral ethmoid and right maxillary sinusitis.

Examination of the nose and throat showed a mucopurulent discharge, which was visible also in the nasal pharynx. Enlarged retropharyngeal lymph nodes could be seen on both sides. The tonsils had been removed. There was, however, a fairly large adenoid rest. On January 22, I punctured and irrigated both antrums, and evacuated mucopus from both sides. On February 2, both sides of the nose and throat appeared free from pus. I then removed the adenoid rest. Since then, the patient has developed uninterruptedly and is in excellent condition. He has not had any recurrence of the fevers.

CASE 4.—*Intermittent fever following influenza.*

M. W., aged 9, came to my office on Oct. 3, 1926, because of intermittent fever for the last two months, following an attack of influenza.

Nasal examination revealed pus in the left side of the nose. X-ray examination showed a distinct ethmoiditis and maxillary sinusitis on the left side. Irrigation of the left maxillary sinus gave prompt relief from the fever.

CASE 5.—*Repeated infection of the middle ears.*

A. R., aged 7, was brought to my office on June 28, 1925, because of repeated abscesses of the middle ear, profuse nasal discharge, particularly from the right side, poor appetite and occasional cough. She had had the tonsils and adenoids removed at the age of 1½ years, and had had two subsequent operations on the adenoids without improvement. It was stated that little was found at the last

Fig. 5.—Right ethmoiditis.

operation on the adenoids. Conservative nasal treatment, followed by nasal irrigation with a 1 to 1000 solution of mercurochrome gave rapid relief. She remained free from symptoms until March, 1926, when she developed an acute cold and ran a temperature fluctuating between 99 and 100 F. for about three weeks. Following two nasal treatments, the fever subsided, and she has remained well since.

CASE 6.—*Sneezing, nasal obstruction and discharge.*

J. E., aged 10½, came to my office on Aug. 27, 1927, complaining of sneezing, nasal obstruction and nasal discharge. She suffered from repeated infections of the ears, and frequent irregular rises in temperature, ranging at times to 105 F. with apparently slight colds. Headaches were not present.

Nasal examination showed mucopus in the right side of the nose. The x-ray showed distinct ethmoiditis on the right side. Packing with mild silver protein, suction and nasal irrigation promptly relieved all symptoms.

CASE 7.—*Listlessness and headache.*

A. S., aged 7, came to me on Nov. 8, 1926, suffering from general tiredness, listlessness and headaches.

Nasal examination showed mucopus in the left side of the nose. The x-ray revealed a positive condition in the antrum. The patient improved considerably under local treatment; the parents, however, would not allow puncture of the antrum.

Fig. 6.—Bilateral maxillary sinusitis.

CASE 8.—*Acute otitis with acute sinusitis.*

R. L., aged 8, seen on June 8, 1927, had an acute otitis media on the right side, for which I performed a paracentesis. The nose showed a distinct purulent discharge on the right side. X-ray examination of the sinuses showed ethmoiditis and maxillary sinusitis on the right side. The nasal condition improved under local therapy.

This case is reported to indicate the simultaneous involvement of the accessory air spaces of the nose and ear. In acute cases, these involvements of the sinus will resolve in the same manner as an acute otitis simplex.

CASE 9.—*Headaches, vertigo and loss of appetite.*

R. M., aged 9½, came to me on April 21, 1928, complaining of right frontal headaches, following influenza one month before. The patient suffered from occasional vertigo and loss of appetite. He appeared fidgety and nervous, and had a hacking cough and heaviness in breathing at night. His complexion was pasty, and there was a distinct puffiness of the lower lids. He suffered from a moderate degree of constipation, and complained of pain in the arms and legs.

Nasal examination revealed distinct evidences of pus in the right middle meatus, as well as some infected tonsil rests. X-ray examination revealed a

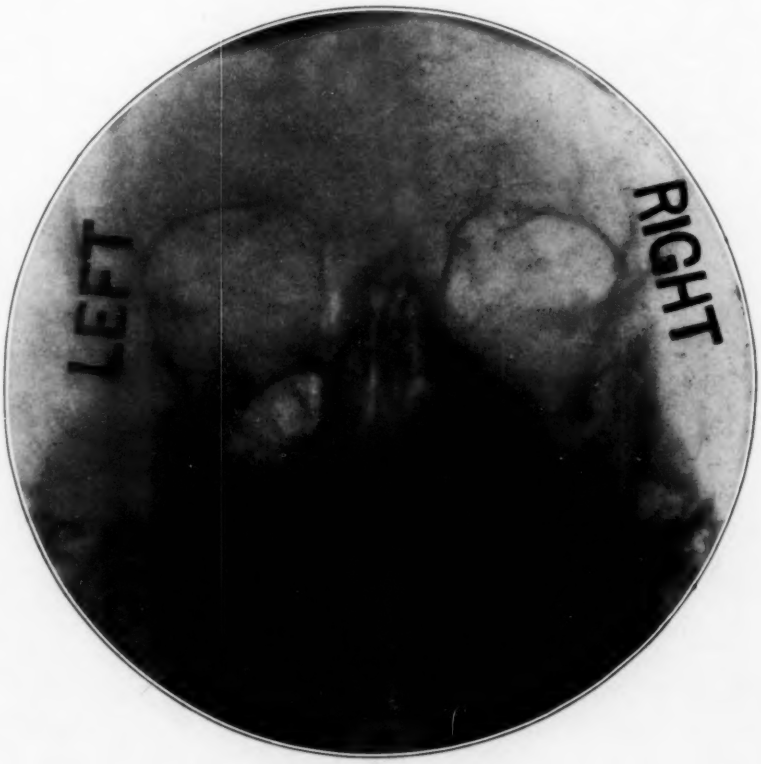

Fig. 7.—Right maxillary sinusitis.

distinct ethmoidal and maxillary sinusitis on the right side. Under local anesthesia, I punctured and irrigated his right maxillary sinus, and evacuated a considerable amount of thick, greenish pus. After three irrigations, his general condition had improved splendidly. The headaches disappeared, and the nasal discharge had ceased. I subsequently also removed the infected tonsil rests.

CASE 10.—*Nasal obstruction with profuse discharge.*

C. G., aged 8, came to my office for treatment on Jan. 26, 1927, because of nasal obstruction, with profuse nasal discharge, and redness and superficial erosions on the alae of both nostrils.

X-ray examination showed bilateral ethmoiditis which responded to local treatment and nasal irrigation.

CASE 11.—*Headache and nasal discharge from both sides.*

M. F., aged 7, appeared for examination on June 14, 1928, suffering from headaches across the forehead, and nasal discharge from both sides. She had had repeated attacks of grip with the temperature ranging around 100 F. She suffered from a slight cough, nervous irritability and insomnia, and for the last year had gained nothing in weight. One year before, she had her tonsils and adenoids removed and since that time had felt rather worse than before.

X-ray examination revealed a bilateral ethmoiditis and maxillary sinusitis. I punctured and irrigated both antrums, and evacuated thick pus from both sides. Her general condition has been improving remarkably.

Fig. 8.—Right pansinusitis.

CASE 12.—*Pyelitis, mastoiditis with recurrent otitis of the middle ear, secondary to suppurative sinusitis.*

E. K., aged 3½ years, came to my office with a history of having taken sick five weeks before with an acute nasal cold. A week and a half later, the patient developed otitis media on the right side, for which a paracentesis was performed; one week later paracentesis was required in the left ear. At the same time, the child began to have pus cells in the urine, and she developed a pasty complexion, with puffiness under the eyes. She had had a continuous fever, the temperature ranging between 100 and 103 F. for the five weeks prior to my seeing her.

Examination showed that the child looked rather chronically ill, although immediately prior to the last illness she had been robust. Nasal examination revealed a mucopurulent discharge in both sides of the nose. Both middle ears showed a thick, yellow discharge. There was not any tenderness in the mastoid and only a slight sinking of the posterior part of the canal wall. The patient was hospitalized on March 29, 1928, and was kept under observation for a period of two weeks at the New York Post-Graduate Hospital. Her condition during this time remained practically stationary, the ears discharging profusely and the temperature continuing to be irregular. The child appeared well and was apparently comfortable. An x-ray picture of the mastoids on her admission to the

Fig. 9.—Bilateral ethmoiditis.

hospital showed a slight clouding. Two weeks later, the x-ray picture showed only a slight increase in the haziness of the mastoids, with some slight disappearance of the bony septums. The urine during this time showed pus cells intermittently. There was still practically no sinking of the canal wall, no mastoid tenderness and no retro-auricular thickening. The appetite of the child, however, was diminishing, and the discharge from the ears continued about the same as when she was admitted. After considerable deliberation, a double mastoid operation was decided on. Both mastoids showed an extensive coalescent mastoiditis with little involvement of the cortex, but considerable destruction of the mastoid cells. The mastoid wounds healed rapidly and both middle ears were dry after

the first week. On May 18, the mastoid wounds and middle ears were healed. The nasal condition also subsided. On May 20, the child appeared again with a nasal discharge and an acute otitis media on the right side.

X-ray examination revealed a distinctly cloudy right antrum. The right antrum was punctured and irrigated with a 1:5 solution of hexylresorcinol, and a thick, greenish-brown mucopus was evacuated. The right ear was dry on the third day, and the child began to improve remarkably. Within two weeks, she had gained weight and developed a robust appearance.

In this entire case, I have the feeling that the sinusitis had caused both the pyelitis and the mastoiditis, and that the neglect of the sinuses might have led to a chronic suppurating ear. This observation has been borne out in many of my cases, and I, therefore, feel that every case of chronic otitis in childhood should be studied with the aid of a roentgenogram of the accessory nasal sinuses.

351 West Eighty-Sixth Street.

FELIX PLATTER

1536-1614

JOHN RUHRÄH
BALTIMORE

Felix Platter was born in Sion. His father was a learned scholar, but not much of a business man. In spite of the financial condition of the family, Felix was sent to Montpellier, where, after five years of assiduous study, he received his doctor's degree in 1557. Following this, he and his brother Thomas traveled about France, and after his *"Wanderjahr,"* which included part of Germany, he returned to Basel, where he lived a long and useful life. For fifty-four years, he was archiater and professor of medicine. He founded a botanical garden, which, as he was childless, he willed to his students. He had a marvellous collection pertaining to natural history and was an enthusiastic anatomist. He was the first of the new Vesalian school in Germany and is said to have made the first dissection of the human body in that country. During his life, his dissections ran into many hundreds.

Platter was a practitioner of great renown; his advice was much sought after, and he was much in demand for consultations. As a teacher, he was no less successful, and his lecture hall was crowded for half a century with students from all over Europe. When the plague devastated Basel, he remained at his post and rendered wonderful service. He was beloved by his patients and respected and honored by all.

He was a prolific writer, covering a wide range of subjects, but his best known publications deal with anatomy. His "De Corporis humani structura et usu libri III" was illustrated with plates from Vesalius and Coiter, but the plates on the eye and ear were of his own making. He also wrote a work on the female genitalia. His book on practice, in three volumes, was immensly popular and went through many editions. A charming little book is the account of the wanderings of his brother and himself as students. This is in German, but has also been translated into French, and it will repay perusal.

Platter's pediatric contribution, while short, is of the greatest importance. It contains presumably the first description of death caused by enlargement of the thymus. It is included in his "Observationum in hominis affectibus plerisque," Basel, 1614. The entire quotation is as follows:

Suffocatio a struma interna abscondita, circa jugulum. Filius Marci Peresii, quinque menses natus, bene habitus, nullo precedente alio affectu, subito cum stridore et respirationis difficultate e medio sublatus est; quo genete mortis prius

FELIX PLATTER
1536-1614

quoque cum duos amisisset filive, causam scire cupiens, ipso rogante, pectus aperuimus, et in regione juguli, glandulam illius locii in struman quondam, magnam, unciam unam cum semisse pendentem, spongiosam, venisque refertain, et quae beneficio membranarum duntaxat, vasis maxomis illis juxta jugulum ascendentibus, adhaerebat, excrevisse, deprehendimus, carniformi quadam materia et sanguine oppleta, quo subito irruente strumamque illam delatante, adeo ut vasa illius loci premeret, suffocatum fuisse infantum judicavimus.

[Suffocation from a hidden internal struma, about the throat. The son of Marcus Peresius, five months of age, well nourished, with no previous illness, suddenly died from difficult breathing, suffocation. As the father had previously lost two sons from the same malady and being desirous of knowing the cause, we opened the chest, at his request. We found the gland in the region of the throat as a large protruding tumor, one ounce in weight, spongy, fleshy and pendent, replete with veins, adhering by membranes to the largest ascending vessels adjacent to the throat; these being filled with blood and flowing into the struma, dilated it to such an extent that it compressed the blood vessels in the locality; in which manner I concluded the child was thus suffocated.]

News and Comment

UNIVERSITY NEWS

New Pediatric Department at Marquette University.—Marquette University Medical School, Milwaukee, has organized a separate department of pediatrics. The following appointments have been made: M. G. Peterman, M.D., head of the department; A. B. Schwartz, M.D., clinical professor; A. L. Kastner, M.D., associate professor; K. E. Kassowitz, M.D., assistant professor; S. H. Lippitt, M.D., assistant clinical professor; H. O. McMahon, M.D., assistant clinical professor; G. F. Kelly, M.D., and F. R. Janney, M.D., instructors, and S. E. Kohn, M.D., assistant.

WORK OF CALIFORNIA INSTITUTE OF CHILD WELFARE

The University of California Institute of Child Welfare will make an intensive study of the development and growth of 150 infants. It is planned to obtain roentgenograms of the development of the normal chest and lungs, and if possible, to detect how early changes in the lymph glands begin in infants who are tuberculous.

DEDICATION OF NEW YORK MEDICAL CENTER

On Oct. 12, 1928, the entire Medical Center of New York City was dedicated to its threefold purpose of teaching, research and care of the sick. The Babies Hospital in the City of New York, which will be completed and occupied shortly after the first of the year, was one of the units dedicated. It has accommodations for 80 small children and 118 larger children.

DEATH OF DR. GARCIA JUST AFTER AWARD OF PRIZE

The Mitre Institution of Buenos Aires awarded the first prize of 2,500 pesos to the late Dr. Carlos Lagos Garcia for clinical study on pulmonary hydatid cysts in children (120 observations). Dr. Garcia died a few hours after the board awarded the prize.

Abstracts from Current Literature

Anatomy and Physiology

ATROPHY OF THE PAROTID SALIVARY GLAND. JAMES MAXWELL, Proc. Roy. Soc. Med. **21**:1249 (May) 1928.

The patient was first seen in the hospital in October, 1921, suffering from an enlarged and tender gland below the left mastoid process near a sinus which was at the site of an incision made into an abscess five weeks previously. The diagnosis of tuberculous glands was made at this time, and the boy was placed on general treatment for a few months, without improvement. X-ray treatment was then given, after which the patient was lost sight of for three and a half years. At the end of this time, he was undersized and underweight, and thyroid extract was given on the assumption that the x-ray treatment might have inhibited normal growth through the thyroid gland. A year later he had mumps. When he was seen the next year, facial asymmetry was noticed. The lower and posterior part of the parotid gland was completely atrophied, with a resultant hollowing of the cheek. A flow of saliva had not been observed from the left Stenson's duct. There were two possible causative factors: first, the x-ray treatment and second, the mumps.

WILLIAMSON, New Orleans.

ISO-AGGLUTINATION AND THE BLOOD GROUPS IN THE COURSE OF INFANCY. ROBERT DEBRE and MAURICE HAMBURGER, Rev. franç. de pediat. **4**:127, 1928.

The properties of the blood having to do with the phenomenon of agglutination were studied in more than 100 infants ranging in age from birth to 2 years. It was found that the blood group of the infant could be determined at any time by checking the corpuscles against serums of group II and group III (the Beth Vincent method). But although the agglutinogens of the corpuscles are present at birth and one may predict what agglutinins the serum will contain, the latter are not always present at birth, and appear to increase in strength only gradually with increasing age. The usual weakness of the agglutinins in early infancy explains how successful transfusions have been possible without previous matching of the bloods, although such a procedure is always deplorable. In almost all cases, the agglutinins are present by the end of the second year. There were four infants whose corpuscles contained neither agglutinogen *a* or *b*, whose serum, instead of developing both agglutinins *A* or *B*, as is typical of group IV, contained only one or the other of the agglutinins.

In 40 per cent of the cases, the blood group of the infant was not the same as that of the mother and in 25 per cent it was incompatible. The properties having to do with agglutination are hereditary and follow the mendelian law.

LESLIE, Evanston.

ACIDOSIS AFTER EXERCISE. H. SALVESEN, Norsk. Mag. f. Laegevidensk. **89**:121, 1928.

The author has found a diminished alkali reserve in persons in more or less good training after a run of 100, 400, 1,500 and 10,000 meters. The highest acidosis was found after the 1,500 meter run, when the lowering of the alkali reserve amounted to 30 per cent by volume. A relationship was not found between the degree of acidosis and the degree of training present. After a run, a large amount of lactic acid is circulating in the organism. A small amount of this acid is excreted in the urine during and after the run. After the 10,000 meter run even less organic acid was excreted than in the control period.

TOVERUD, Oslo.

Pathology, Bacteriology and Biochemistry

THE RELATION OF PARATHYROIDECTOMY TO PREGNANCY, LACTATION AND THE GROWTH OF OFFSPRING IN ALBINO RATS. S. B. CHANDLER, Anat. Record **35**:7, 1927.

The parathyroid glands were removed from albino rats between the ages of 75 and 100 days; the animals were given from 2 to 3 weeks to recover from the effects of the operation and then were mated with both normal and parathyroidectomized animals. A standard diet was used, with a liberal supply of meat every other day. Ten litters were secured. One female died 23 days after mating. She had seven fetuses near term. The offspring of four other females died soon after birth and were eaten. The mothers apparently were unable to care for their young in these four cases. The weight of many of the offspring at birth was a little less than the normal, being about 4 Gm. in most litters. Growth was much retarded in some litters.

KUGELMASS, New York.

PATHOLOGIC ANATOMY OF THE BRAIN IN MYXEDEMATOUS CHILDREN. L. KENA-APAJALAHTI, Acta Paediat. **7**:42 (April) 1928.

The brain of a cretin who died of bronchopneumonia at the age of 13 months, after having been under treatment since the age of 8½ months, showed at autopsy a diminution in size, particularly of the frontal lobe, a decrease in the cells of the molecular layer and particularly in the middle pyramidal cell zone, and loculi in the basal ganglions and pons. The author advances the opinion that a congenital malformation of the brain may accompany congenital absence of thyroid tissue and thus explain the frequently disappointing results of thyroid therapy in promoting normal development of the psyche.

CHANGES IN THE REACTION OF THE BLOOD AS A CAUSE OF DISEASE. PAUL DRUCKER, Acta Paediat. **7**:220 (April) 1928.

Drucker believes not only that considerable variations in the p_H of the blood are found as an accompaniment of certain disease conditions, but that such variations are of secondary importance in symptomatology. By administering fairly large doses of ammonium chloride (1 Gm. four times a day) to healthy infants, he was able to induce a depression of as much as 0.37 p_H without in any way giving rise to detectable symptoms of acidosis.

SEASONAL VARIATIONS IN THE PHOSPHATE CONTENT OF THE BLOOD DURING THE FIRST TWO YEARS OF LIFE. KARIN BRUUN, Acta Paediat. **7**:226 (April) 1928.

Monthly determinations of the inorganic blood phosphate were made in five infants throughout one year. Two of these were new-born infants, in whom the first determination of phosphate showed a value of less than 3 mg. in 100 cc., after which the level rose to the vicinity of 5 mg. Aside from this physiologic change, previously described, there was no regular variation in the phosphate level depending on the age of the patient; but in all the patients examined there occurred a pronounced sag in the curve during the first three or four months of the calendar year. These seasonal changes were more conspicuous than those reported by Hess for New York, and may be ascribed to differences in latitude between Helsingfors and New York.

REGULATION OF BLOOD SUGAR IN INFANTS WITH EXUDATIVE DIATHESIS. C. W. HERLITZ, Acta Paediat. **7**:286 (April) 1928.

Using the Hagedorn-Jensen method, Herlitz studied the fasting blood sugar level of normal infants and infants with exudative diathesis, and their response to oral and subcutaneous administration of dextrose. The fasting level of the

latter group was definitely elevated above that of the former. After the administration by mouth of 1.5 Gm. of dextrose per kilogram of body weight in 10 per cent solution, the eczematous infants whose eruption had been present for less than two weeks showed a diminished tolerance, while that of infants whose skin lesions had been present more than two weeks closely followed the normal curve. Similar results were obtained when the dextrose was given subcutaneously in doses of 0.3 Gm. per kilogram in 10 per cent solution. After disappearance of the eruption, the blood sugar curves of the patients with exudative diathesis which had previously been found elevated returned once more to normal.

McIntosh, Baltimore.

Nutrition

Infant Feeding. Résumé for a Period of Twenty Years, Stressing the Importance of Breast Feeding. Coursen B. Conklin, M. J. & Rec. **127**:374 (April 4) 1928.

During twenty years, one has seen many changes in the orthodox modes of caring for the infant's nutrition. The one maxim which has been unchanging is that the ideal food is mother's milk, and until the eighteenth century, breast feeding was the accepted method of caring for the infant's nutrition. The certain tendencies which deprive the infant of his natural food supply are: (1) sociologic; (2) false instruction given to the mother by well-meaning but poorly advised persons; (3) failure of her physician to become conversant with the essentials of breast feeding and yielding too easily to the plea for artificial feeding; (4) the absorption of advertised material on proprietary foods by the relatives of the infant.

The dependable innovations of artificial feeding may be enumerated thus: (1) the stress of caloric value of the food by the pediatricians of today; (2) the addition of an acid to cow's milk so that more concentrated mixtures may be given; (3) the development of dried milks as a replacement for condensed milk; (4) the recognition of the importance of the presence of fat-soluble A, water-soluble B and the antiscorbutic and antirachitic vitamins in the food.

Diarrheal Diseases of Infancy. Harry S. Snyderman, M. J. & Rec. **127**:379 (April 4) 1928.

The form of diarrhea in which the author is primarily interested is that commonly known as cholera infantum or summer diarrhea. He prefers the term alimentary disturbance. This disturbance may be due to food or to bacteria and, according to cause, it is called either a noninfectious or an infectious diarrhea. Excessive acidity is the most common occurrence in the diarrheas and indicates withdrawal of fats and sugar. This principle holds good in both forms. The treatment for the two types differs little.

The child should be starved for from eighteen to twenty-four hours and then given an easily digested protein. Castor oil should be given first; if vomiting is present, lavage is used first and is followed by castor oil. To prevent dehydration or acidosis, physiologic sodium chloride solution should be given by hypodermoclysis or intraperitoneally. If blood is present, irrigation with tannic acid (half per cent) should be given.

The author feeds skimmed milk sweetened with saccharine to which he adds casec. Later, carbohydrates are gradually introduced.

Williamson, New Orleans.

Researches in Lactation. Margaret Frances Lowenfeld, J. Obst. & Gynec. Brit. Emp. **35**:114 (Spring) 1928.

The development of lactation in different women varies considerably in both the rate of appearance of the fluid and in the composition of the milk secreted.

Two main types of development can be distinguished. In the first, the breast shows a tendency to activity during the later weeks of pregnancy; the milk comes in early, is not viscid, is rarely deeper in color than primrose yellow, and is homogeneous; it is obtainable early in definite amounts. Protein and probably ash contents are low. The calcium content remains unchanged. In the second type, the breast is inactive during pregnancy and does not produce an appreciable secretion till after the first twenty-four hours after delivery; the milk is viscid, variable in color, not homogeneous and is obtainable in small quantities. The protein and ash contents are high. The line between these two types is not absolute, and cases were found of multiparas and of primiparas which resembled rather more the group other than their own.

The duration of the presence of colostrum depends on a balance of factors between mother and infant, i. e., on the type of development present and on the vigor of sucking of the infant. Small quantities of early milk have a food value approximating larger quantities of mature milk. The percentages of sugar and protein vary slightly at the beginning and end of a feeding, but the differences are without clinical significance. The time during the feeding when the sample is taken and the method of extraction of the milk do not affect the percentage of calcium and ash resulting in the milk. The percentage of fat in the milk is dependent inversely on the amount of fluid present at the time of extraction, and directly on the amount of pressure exerted on the areola.

Morbid states of the breast appear to be correlated with disturbances in the sugar content of the milk. Cases can, however, occur with profound modification of the sugar content, without any corresponding morbid state in the mother or in the infant.

ADAIR and TIBER, Minneapolis.

HIRSCHSPRUNG'S DISEASE WITH FECAL INCONTINENCE. BERNARD SCHLESINGER, Proc. Roy. Soc. Med. **21**:781 (March) 1928.

Hirschprung's disease was reported in a boy, aged 12 years.

WILLIAMSON, New Orleans.

MILKLESS DIET IN INFANCY. G. ABRAHAM, Arch. f. Kinderh. **84**:36 (April 27) 1928.

The milkless diet consisted of zwieback or meat pudding. Infants fed for more than one year on this diet (milkless) did not differ in any way from children receiving a normal diet. Their stools were acid, and there was a tendency toward alteration of the stool flora toward the gram-positive side. The number of colon bacilli diminished markedly.

THE AMOUNT OF ANTINEURITIC VITAMIN B IN COW'S MILK. P. REYHER, Arch. f. Kinderh. **84**:55 (April 27) 1928.

The author found that in Berlin cow's milk was not only low in vitamin C, but in vitamin B as well. In winter, milk was lower in this vitamin, as a general rule, than in summer. This would make it possible that artificially fed infants may suffer in the winter from a lack of vitamin B. This vitamin is essential. It not only affects the nervous system, but offers a resistance to infectious diseases and stimulates the appetite.

SANFORD, Chicago.

THERAPY WITH ERGOSTEROL. F. HOLTZ, Deutsche med. Wchnschr. **53**:706, 1927.

Rachitic rats treated with 0.01 mg. of irradiated ergosterol in paraffin oil or olive oil were healed in from two to three weeks. The healing was manifest through an increase in the acid-soluble serum phosphorus concentration and the definite calcification in the cartilage. Rats that were fed on a rachitogenic diet, to which was added from 0.001 to 0.002 mg. of irradiated ergosterol per day, failed to develop the disease.

ERGOSTEROL IN RICKETS. BEUMER and FALKENHEIM, Deutsche med. Wchnschr. **53**:1115, 1927.

The authors reviewed the history of the discovery of the provitamin of the antirachitic factor and also of the effect of irradiated ergosterol on infantile rickets. They irradiated ergosterol either in alcoholic solution or in triolein for forty-five minutes. The altered provitamin in its active condition lost the property of forming an insoluble digitonin compound. After the administration of from 2 to 3 mg. daily, there was rapid and obvious healing of the rickets in its chemical, roentgenographic and clinical aspects. Increase of the serum phosphorus to the normal usually occurred in ten days. Only one patient who had febrile complications (lymphadenitis) did not show a change in the blood chemistry but did show an increased deposition of calcium roentgenologically.

YOLK OF EGG IRRADIATED WITH ULTRAVIOLET LIGHT. F. ROHR and O. SCHULTZ, Deutsche med. Wchnschr. **53**:1255, 1927.

The authors attempted to enhance the antirachitic value of egg yolk by exposing it to ultraviolet light. They failed in their attempts, but the irradiation did not destroy the antirachitic potency of the egg yolk.

IRRADIATED ERGOSTEROL IN RICKETS OF ANIMALS, CHILDREN AND ADULTS. A. HOTTINGER, Deutsche med. Wchnschr. **53**:1549, 1927.

The author advises a dose of 1 mg. of irradiated ergosterol a day for children under 1 year of age and from 2 to 3 mg. for children over 1 year with severe rickets. Children with florid rickets were cured clinically, chemically and radiographically in from three to four weeks.

IRRADIATED ERGOSTEROL IN CHILDREN. W. LASCH and A. BEHRENS, Deutsche med. Wchnschr. **53**:1552, 1927.

The authors studied five infants whose ages ranged from 6 to 18 months. The infants were in an infant asylum and developed rickets while under observation. They were given from 3 to 4 mg. of irradiated ergosterol which in some instances was dissolved in oil and in others was administered in the form of a chocolate tablet. The first effect of the treatment was a definite change in the infants' behavior. The children became happier, more active, began to coo and showed a definite interest in their environment. They soon began to raise their heads and the upper part of the body. Craniotabes which was present in the five children, disappeared in every one in fourteen days. The roentgenograms showed definite fresh deposition of lime salts in the bones at the end of two and one-half weeks. The authors also carried out prophylactic studies. In their experience, premature infants and twins, even though breast fed, invariably developed rickets in the first months of life even during the summer. They attempted to find whether the prophylactic administration of irradiated ergosterol would prevent the development of rickets in this type of infant. They used two pairs of twins, who shortly after their birth were given 3 mg. of ergosterol daily along with breast milk. Only one developed a minute area of softening in the partietal bone. But the calcium and inorganic phosphorus concentration in the serums was normal. The authors did not consider these areas as true craniotabes. These infants gained on an average of from 910 to 990 Gm. every month. The authors expressed the belief that the antirachitic effect of ergosterol is comparable in its speed and intensity to ultraviolet light from the mercury vapor quartz lamp.

IRRADIATED ERGOSTEROL (VIGANTOL); CLINICAL AND EXPERIMENTAL OBSERVATIONS. H. VOLLMER, Deutsche med. Wchnschr. **53**:1634, 1927.

Vollmer studied the effect of irradiated ergosterol and found that the increase of the inorganic phosphorus of the serum does not necessarily precede the deposition of lime salts in the provisional zone, but that such deposition may occur even with

subnormal concentration of phosphorus in blood serum. As a suitable dose they used 5 mg., that is, 0.5 cc. of a 1 per cent solution of oil. More rapid healing occurs when the dose is increased to 10 mg. The first evidence of calcium deposition in the bone appears after nine days. Occasionally at the end of treatment, abnormally high values for serum phosphorus were obtained. When large single doses of irradiated ergosterol were given to children, that is, 120 mg., the results were not as favorable as after the repeated administration of small doses. Irradiated ergosterol in olive oil or in ether was injected subcutaneously, but the therapeutic effect was protracted so that the authors returned to the oral administration. These experiments, however, show that irradiated ergosterol is effective when injected parenterally. Five tenths milligram of irradiated ergosterol did not suffice to protect infants against rickets, but 1 mg. daily was effective.

KRAMER, Brooklyn.

OBSTIPATION IN CHILDREN. FRANZ HAMBURGER, München. med. Wchnschr. **75**: 1210 (July 13) 1928.

Obstipation must be divided into the acute and chronic forms, of which the acute are dangerous. The various types are described and treatments outlined.

In regard to the chronic constipation of children, attaching too much importance to having a daily stool at a certain time of the day is emphasized and advised against. Even if a child does not have a stool for one or two days it is no misfortune. Selection of a diet and drinking of 100 cc. of warm water early in the morning are regarded as of importance.

SCHORER, Kansas City, Mo.

SILO MILK. ELSE ZOELLER, Ztschr. f. Kinderh. **44**:517 (Nov.) 1927.

The rickets produced in a few weeks in rats by the low phosphorus Sherman-Pappenheimer diet no. 84, can be readily prevented by the addition to it of 2.5 cc. of raw milk. This antirachitic property is possessed, it is shown, by milk obtained from cows fed or not fed with a green food which has been preserved in a silo (silo milk). The addition of such a small amount of raw milk also guarantees a normal gain in weight and growth. If the essential factor $\frac{Ca}{P}$ of the diet is approximated by the enrichment of milk calcium, a definite hypophosphatemia results, but clinical or roentgenologic signs of rickets do not appear.

If the milk fortified with calcium is used after cooking, even with the feeding of 5 cc. of milk, the phosphorus value of the plasma is uniformly diminished, but definite skeletal changes are shown only by the rats who are fed 2.5 cc. of milk. Whether the milk fed is silo milk or ordinary milk does not make any difference. The gain in weight of all the animals, outside of physiologic variations, was small. The therapeutic use for fourteen days of small quantities of either raw silo milk or ordinary milk in rats 5 months old, fed for fourteen days on diet no. 84, did not succeed in curing those with phosphorus-poor rickets. With 2.5 or 5 cc. of raw milk, there was a barely recognizable increase in weight. On the basis of clinical experience in infants, silo milk did not appear to possess any special substances protective against rickets. The slight tendency to healing noticed in three cases could be ascribed to better nursing care. Infants could be safely fed on silo milk, but this is not advisable. Four of six children became ill directly after a short use of silo milk. Cooked silo milk was well borne.

EMERSON, Boston.

THE INFLUENCE OF FOOD AND OF THE EXTERNAL WORLD ON GROWING ANIMALS. R. BAMBERGER, Ztschr. f. Kinderh. **44**:556 (Nov.) 1927.

Researches were made by Bamberger on the relation of diet fat to depot and cell fat to see whether, teleogically considered, nature gives to the rapidly growing suckling a natural food from the beginning, partly because quickly growing tissue is unable to defend itself against unaccustomed quality and quantity in a foreign food and partly because, in a foreign food as in a natural one, the composition of

the body, analogous to the dictum of Bunge, assumes the features of the food given. A new method of fat extraction from body tissues, which keeps the chemical structure of the fat unchanged, was worked out for the solving of this problem, and the quantitative errors reduced to 2 per cent as against from 20 to 30 per cent in the old methods. A new method is given for the rational characterization of fats by means of numbers. It was established that in the feeding of different fats or different carbohydrate or protein mixtures, under different conditions of the external world, the quality of the depot and cell fat in purely bred, related animals differed regularly. As the most important factor of influence for the quality of the cell fat was the content of the diet fat in volatile water-insoluble fatty acids, it was a matter of indifference whether the feeding of higher quantities of water-insoluble fatty acids was combined with much protein or much carbohydrate; or under what conditions of the external world the animals grew. When higher quantities of such fatty acid were fed, higher quantities of them were found in the depot and cell fat of all systems of organs.

The analytic observations in both animals of the experiment which did not thrive were outside the limits of the observations of the twelve animals which did thrive and varied to a much greater degree than is referable to the law of probability.

EMERSON, Boston.

WEIGHT DEVELOPMENT IN BREAST-FED INFANTS DURING THE FIRST TWENTY-SIX WEEKS. CARL SCHIÖTZ and H. BERGHOFF, Acta Paediat. **7**:216 (Feb. 28) 1928.

Schiötz and Berghoff have compiled tables of standards, mathematically treated, from the poorer classes of Norway.

A, B AND C VITAMIN CONTENTS OF COD LIVER OIL. ERIK AGDUHR, Acta Paediat. **7**:289 (Feb. 28) 1928.

Are the so-called "A" vitamins in cod liver oil the cause of its toxic effect on the organism; and can a basal diet, complete as regards the so-called "B" and "C" vitamin contents, prevent this toxic effect? Agduhr presents a lengthy rebuttal of this subject.

MCINTOSH, Baltimore.

Prenatal Conditions

FETAL MORTALITIES. GEORGE KAMPERMAN, Am. J. Obst. & Gynec. **16**:66 (July) 1927.

The author has divided the fetal deaths occurring on the obstetric service at the Harper Hospital during 1925-1926 into three groups. The groups include 163 deaths, a fetal mortality of 6.65 per cent.

In the first group are the fetal deaths occurring between five and seven months before term, a total of sixty-three cases.

The second group, eighty-five in number, includes those born prematurely but who were viable.

The last group of seventy-six cases includes all deaths occurring from the beginning of the last month of pregnancy to term.

In all groups, the cases of unknown causation of death comprised the highest percentage—22.7. Nephritic toxemia came next, with 20.2 per cent. Placental bleeding, malformations and death at delivery each caused a death rate of 11 per cent.

Syphilis and pneumonia, on the other hand, caused only one death each, a ratio of 0.6 per cent. Postmaturity is given as the cause of death in 2.4 per cent of the cases, or more than 0.6 per cent more frequent than death from eclampsia. This speaks well for the prenatal care that these patients have had.

ALLEN, Chicago.

Diseases of the New-Born

TUMOR OF SACROCOCCYGEAL REGION IN THE NEW-BORN. M. HERZFELD, Edinburgh M. J., Trans. Obst. Soc. **35**:89 (June) 1928.

Miss Herzfeld reported an infant born with a tumor of the sacrococcygeal region as large as the infant's head. Within a few hours, the thin overlying skin showed evidence of necrosis. Operation was at once performed, and this sessile growth, which had been palpable through the rectum and had displaced the rectum anteriorly, was removed. Local anesthesia permitted an easy removal, though the tumor was adherent to the rectum and the levators of the anus were much separated. Recovery was complete. The tumor was partly cystic and solid, and the diagnosis lay between teratoma and a growth from the body of the coccyx, the nature of which is to be reported later.

NEFF, Kansas City.

Acute Infections

A STUDY OF THE VALUE OF CONVALESCENT'S SERUM IN THE PREVENTION OF MEASLES. A. J. SCHAFFER, Buffalo Gen. Hosp. Bull. **6**:14 (July) 1928.

About 100 infants and children exposed to measles were given convalescent's serum or whole blood. Four of these developed measles. Of these four, only one received serum one day after exposure; the others had received doses thirty-two, thirty-four and seventy days previous to their last known exposure. In a control group of approximately thirty infants, six developed the disease.

SANFORD, Chicago.

SPECIFIC PREVENTION AND TREATMENT OF EPIDEMIC POLIOMYELITIS. SIMON FLEXNER and FRED W. STEWART, New England J. Med. **199**:213 (Aug. 2) 1928.

It is an established fact that in a person who has recovered from poliomyelitis the blood contains virucidal or neutralizing antibodies for the virus of the disease. This fact is equally true of man and the monkey. An attempt has not yet been made to induce active immunity in man by inoculation of the virus; but an active immunity of this kind has been produced in the monkey by repeated injection of small quantities of the virus. Actively immunized monkeys are protected against intracerebral injection of the virus, and their blood carries the virucidal antibodies. No other experimental animal has shown itself susceptible to inoculation with the virus or has developed specific antibodies on repeated injections of virus-containing nervous tissues. Of the serums of domestic animals tested, sheep serum alone was found to possess the power in certain instances of inhibiting the action of the virus on monkeys. This natural inhibiting effect is not increased, apparently, by repeated injections of the virus.

The employment of convalescent serum therapeutically is recommended, and the method of use in favor is the combined intrameningeal and intravenous injection of human convalescent serum. The two injections are made at practically the same time, the larger dose, of course, being given intravenously. This injection is not repeated, although the intraspinal injections are repeated two or three times on successive days.

In regard to the use of other serums, the authors say that it is important to note that convalescent serum is demonstrably effective in the monkey—the basis of its employment in man—while the antistreptococci serum is demonstrably ineffective in this animal which alone is susceptible to the experimental disease.

The authors have recently tested the duration of an intraspinal injection of convalescent serum on a subsequent intracerebral inoculation of virus. The quantity of convalescent serum injected by lumbar puncture into monkeys was small (2 cc.), in spite of which an intracerebral injection of virus was rendered ineffective four days, but not six days, after the intrameningeal serum was introduced.

Flexner and Amoss have shown that large intravenous doses of the virus may be ineffective in monkeys when the meningeal choroid plexus apparatus is intact. They also showed that as long as this intactness continues, immune serum does not pass from the blood into the cerebrospinal fluid. They ascertained that this state of intactness is easily disturbed and that when it is disturbed, much smaller quantities of virus injected into the blood will induce paralysis and also that virucidal antibodies pass into the cerebrospinal fluid.

Now in poliomyelitis in man, even in the mild, recognizable forms, the meningeal choroid plexus apparatus is not intact, a fact shown by the increase in cellular or protein content, or both, of this fluid. When an intracerebral inoculation of the virus is made in monkeys, not only is a lesion produced in the brain at the site of injection, but the virus-carrying salt solution floods the meninges and itself produces mild chemical injury, which alter the intactness.

If as much as 15 cc. of the convalescent serum is injected into the blood of monkeys twenty-four hours before a suitable dose of filtered virus is injected intracerebrally, infection does not take place. The test is severe, because a lesion is produced in the cerebral tissue at the site of inoculation, and also a locus of reduced resistance in which the virus rests is thus created. It is even possible that a smaller intravenous injection of serum may protect.

On the basis of the tests which have been briefly outlined, the authors have proposed that convalescent human serum should be employed at times of stress and anxiety, when poliomyelitis is epidemic, for producing passive immunization. The doses of the convalescent serum which they have suggested are 10 cc. for children and 20 cc. for adults, injected subcutaneously and repeated after a period of from four to six weeks, if the danger still continues.

In the passive immunization tests in monkeys, several days—called the incubation period of the disease—elapse between inoculation and the appearance of the first symptoms. This period permits the immune antibodies to become well distributed throughout the central nervous system. In preparalytic or early paralytic poliomyelitis in man, the first effects of the virus have taken place, and the next events may follow quickly. There is therefore not the same period of days or even hours provided for the immune antibodies in the blood to reach in concentration all parts of the nervous organs. Consequently, it would seem advisable, theoretically at least, that one intraspinal injection of the convalescent serum should be given at the earliest practicable moment. This will insure flooding of the nervous tissues with antibodies through intermediation of the cerebrospinal fluid, after which the maintenance of the antibody contact may perhaps be left to the increased permeability of the meningeal choroid plexus barrier to the contents of the circulating blood.

GENGENBACH, Denver.

PITUITARY EXTRACT IN PYELITIS. JULIUS A. MILLER, N. Y. State J. Med. **28**:719 (June 15) 1928.

The author, having found that pyelitis did not occur in obstetric patients when pituitary extract was used, gave the same treatment to other pregnant and nonpregnant women with excellent results. He suggests that similar treatment might be tried by pediatricians. The good results are attributed to the restoration of tone to the ureter and prevention of urinary stasis.

AIKMAN, Rochester, N. Y.

NONSURGICAL SUBCUTANEOUS EMPHYSEMA IN LARYNGEAL DIPHTHERIA. J. D. ROLLESTON, Proc. Roy. Soc. Med. **21**:771 (March) 1928.

A girl, aged 6 years, was admitted to the hospital on October 24, on the fifth day of the disease, with a membrane on the tonsils and pharynx and dyspnea and recession. In spite of three large doses of antitoxin, the dyspnea increased. On October 26, subcutaneous emphysema appeared on the neck and spread up to the face and down to the costal margin. Tracheotomy was performed at

5 p. m. and gave considerable relief, but the emphysema spread. There was no cardiac dulness, and the heart sounds were distant. The emphysema was slow in subsiding and not until the eleventh day had it completely gone. The tube was removed on the ninth day. The child subsequently contracted a mild attack of scarlet fever followed by chickenpox, but otherwise recovery was uneventful. The rarity and etiology of the condition are discussed in the report.

WILLIAMSON, New Orleans.

THE INFLUENCE OF INFECTIONS ON THE DEVELOPMENT OF INFANTS IN INSTITUTIONS. S. LEVY, Arch. f. Kinderh. **84**:27 (April 27) 1928.

Levy compared the amount of weight lost and the time required to regain it in an epidemic of dysentery, with an epidemic of grip enteritis. In the patients with dysentery, the average loss of weight in 4.5 days was 435.5 Gm. Recovery required about 23.5 days, on the average, and it was 58 days before the lost weight was regained. The grip enteritis averaged 437 Gm. in 6.5 days. Recovery took 21 days, with 43.5 days to regain the lost weight.

SANFORD, Chicago.

INVOLVEMENT OF THE LOWER RESPIRATORY TRACT IN SCARLET FEVER. H. DEICHER, S. TANNHAUSER and A. SALINGER, München. med. Wchnschr. **75**:1199 (July 13) 1928.

While involvement of the upper respiratory tract in scarlet fever is generally observed, that of the lower tract is less obvious though it may lead to serious complications.

While the severe reactions in the trachea and larynx have been described, the authors emphasize conditions not observed during the acute onset of the disease because of the toxic symptoms.

These involvements consist of inflammation, with or without formation of membrane, in the larynx, trachea, bronchial tubes, and tissue and pleura of the lungs.

The authors gave diphtheria antitoxin a number of times, to be safe, although they did not wait for the culture report, and even when diphtheria organisms were not found.

In these cases, they feel that the scarlet fever antiserum is of the greatest value. In a number of cases, they resorted to tracheotomy and thoracotomy.

SCHRORER, Kansas City, Mo.

THE INTENSIVE LOCAL TREATMENT OF GONORRHEA IN CHILDREN. F. ALTERTHUM, Ztschr. f. Kinderh. **44**:546 (Nov.) 1927.

In Alterthum's experience over a course of six years, an intensive local treatment of gonorrhea in children with various silver salts in different strengths, mild to strong, by the vagina, urethra and rectum, resulted in shortening considerably the duration of the disease and in minimizing the number of relapses. He had tried all of the other treatments in use up to this time without any noteworthy success. No patient was considered cured unless negative cultures followed local treatment for six weeks; then no treatment at all was given for the next six weeks, followed by three weeks of provocative treatment.

A CASE OF MENINGOCOCCUS SEPTICEMIA WITHOUT MENINGITIS. I. VON LEMMERS-DANFORTH, Ztschr. f. Kinderh. **44**:551 (Nov.) 1927.

Von Lemmers-Danforth describes a case in a girl, aged 2½ years, who was sick with meningococcus septicemia for several months and recovered. Four days before admission she became suddenly ill with fever, vomiting, restlessness, headache, pains on urination and pains in the extremities, but she did not have stiffness of the neck or Kernig's sign and the spinal fluid was normal. On the third day of the disease, she showed a rash on her neck and chest like that of measles. At first she was thought to have typhoid fever, but the blood culture showed meningococci. The treatment consisted of the injection of several kinds of drugs and maternal blood.

EMERSON, Boston.

EPIDEMIC OF SCARLET FEVER AT GRONINGEN. S. P. VAN BUCHEM, Nederl. Tijdschr. v. Geneesk. **72**:328, 1928.

The author describes the symptoms observed in 500 patients with scarlet fever in the hospital for infectious diseases at Groningen during the last one and one-half years. Itching and petechiae were often seen in the beginning of the disease. Symptoms of rheumatic scarlatinosis were noticed in 5 per cent of the cases, chiefly during the first four days of the illness and lymphadenitis in 14.8 per cent, mostly during the first two weeks. Otitis media was seen in 10.4 per cent, and acute glomerular nephritis in 0.8 per cent. Endocarditis was seen rather frequently, e. g., in 1 per cent of the cases, and myocarditis occurred in one patient in the beginning of the scarlet fever. In the fifth and sixth week, "febris causa ignata," explained by Finkelstein as a disturbance of the equilibrium of the autonomic nervous system, was observed rather frequently. Three of the 500 patients died. The Dick test was made 363 times in 300 patients. In accordance with other authors, it was found that the number of positive tests decreased when the test was performed at a later stage of the convalescence. Several times the test was still found positive on the fourteenth and the twenty-second day of the illness, whereas the same patients showed a negative test in the fifth and sixth week.

S. VAN CREVELD, Amsterdam.

ADULT HUMAN SERUM AS TREATMENT FOR WHOOPING COUGH. I. JUNDELL, Acta Paediat. **7**:319 (Feb. 28) 1928.

The author treated twenty-four patients (ten during the incubation period or the catarrhal stage and fourteen during the paroxysmal stage) with maternal or other adult serum given subcutaneously. He did not attempt to obtain "convalescent serum" and used the serum whether or not the person giving it had had whooping cough. He was favorably impressed with his results, even admitting the small number of the patients treated.

MCINTOSH, Baltimore.

INFECTIOUS MONONUCLEOSIS. SALVESEN and MAGNUSSEN, Norsk. Mag. f. Laegevidensk. **88**:189, 1927.

Two cases of infectious mononucleosis (glandular fever) in young men were described. They were both characterized by fever, moderately swollen glands, especially of the neck, sore throat and an increased white blood cell count with an increased percentage of lymphocytes (up to 75 per cent). This disease is probably the same as that described earlier by Pfeiffer as "Drüsenfieber" in children. As far as the first case is concerned, the diagnosis of leukocytosis may be excluded as the blood picture was normal one year after the attack of glandular fever. In the other patient a relative lymphocytosis was present two months after the patient was discharged from the hospital, so that the question of this being a leukemia cured by an intercurrent infection is still open.

In one of the patients the blood was studied following an injection of sterile milk during convalescence. A normal reaction was found (leukocytosis due to increase in the neutrophils).

HERPES ZOSTER; VARICELLA. SOLBERG, Norsk. Mag. f. Laegevidensk. **89**:287, 1928.

A man, aged 40, contracted herpes zoster in the summer of 1926. Three weeks later, chickenpox was diagnosed in his wife, aged 39. She had a generalized exanthema with vesicles in the mouth and pharynx. She had some fever. Chickenpox had not been observed in the narrow mountain valley in which she lived for the last five years. It seems as if herpes zoster is infectious and can give rise to a disease resembling chickenpox, which, however, has nothing to do with the usual infectious chickenpox in children.

TOVERUD, Oslo.

Chronic Infections

TUBERCULOUS OSTEITIS OF THE SKULL. O. L. ADDISON, Proc. Roy. Soc. Med. **21**:782 (March) 1928.

The case reported occurred in a boy, aged 4 years. He was healthy until he was 14 months old. At that time a swelling appeared over the right orbital ridge, and a few weeks later a second similar swelling appeared over the ascending ramus of the mandible on the right side. The x-ray did not show any bony changes corresponding with these tumors. The physical condition of the child was good. A course of x-ray treatment over the right temple was given for five weeks. Two months later, the mass broke down, and on exploratory puncture, sterile pus was obtained. Three months later, x-ray examination of the skull showed multiple areas of absorption in the skull. One of these masses was opened, and two bony sequestrums were removed. The material was injected into a guinea-pig, and six weeks later at postmortem, definite evidence of tuberculosis was found in the guinea-pig. The boy is now apparently well. Roentgenography showed that the skull had healed at all sites.

WILLIAMSON, New Orleans.

SURVEY OF TUBERCULOSIS AMONG SCHOOL CHILDREN IN OSLO. THEODOR FRÖLICH, Acta Paediat. **7**:191 (Feb. 28) 1928.

In 1912, the author examined 2,900 school children in the first grade of the Oslo public schools, almost all of them 7 years of age, and noted the incidence of tuberculous infection, the response to the Pirquet test and the presence of adult tuberculosis in the home. At that time, he found 84 per cent of positive skin tests and 4.3 per cent of clinically manifest cases of tuberculosis.

Thirteen years later, vital statistics of the greater part of the same group were obtained covering 1,830 of those who had had a positive skin test and 332 of those with a negative test. Of the positive reactors, 12.2 per cent had, by the age of 20 years, developed some form of tuberculosis, 4.3 per cent had died, and 2.9 per cent had died of tuberculosis. The mortality was higher for girls, as was also the morbidity of pulmonary forms of the infection. For the negative reactors, the corresponding figures were 9.3 per cent, 3.9 per cent and 3 per cent, with again a greater mortality and morbidity among the girls. Of 310 children from tuberculous homes, 19 per cent eventually developed clinical tuberculosis, and, as would be expected, there was not only a higher morbidity but a higher mortality from these homes. The author is of the opinion that in this type of work the severity of the Pirquet reaction is a guide to the severity of the infection and thus has prognostic value.

MCINTOSH, Baltimore.

Internal Diseases

LEUKOPENIC ANEMIA OF THE REGENERATIVE TYPE DUE TO EXPOSURE TO RADIUM AND MESOTHORIUM: CASE REPORT. GEORGE S. REITTER and HARRISON S. MARTLAND, Am. J. Roentgenol. & Radium Therap. **16**:161 (Aug.) 1926.

A review of the literature shows six fatal cases of aplastic pernicious anemia caused by trade exposure to mesothorium and radium. Radioactive material was found in the lungs, bone and spleen, but none in the liver, heart, gastro-intestinal tract or kidneys. By way of the lungs both radium and mesothorium and active deposit from emanation are deposited and finally stored in the organs of the hemopoietic system. From the constant irradiation of this residue, marked effects are produced directly in the leukoblastic and erythroblastic centers. The mechanism is probably a deposition of microscopic dust on the walls of the lungs, with phagocytosis by the alveolar epithelium and endothelium, and access through lymph sinuses to blood stream organs of the reticulo-endothelial system in the bones acting as a source of beta and alpha radiation directly in the blood-forming centers.

STULIK, Chicago.

THE RELATIONSHIP OF THE WEATHER TO THE INCIDENCE OF PNEUMONIA IN THE CHILDREN'S HOSPITAL, BOSTON. LEWIS WEBB HILL, Boston M. & S. J. **9**:93 (July 21) 1927.

From 1915 to 1925 inclusive, 774 patients with pneumonia were admitted to the hospital. Various factors of the weather during that period were investigated, such as the amount of sunshine, temperature, snowfall and rainfall. Graphic charts show the results of these investigations.

The author's summary shows that:

In a period of 132 consecutive months, there were 13 when pneumonia was usually prevalent in the wards of the Children's Hospital. A large number of these months were unusually warm, with an abnormally high rainfall, and abnormally few hours of sunshine. There were 12 months when the incidence of pneumonia was unusually low. Many of these months showed abnormally high amounts of sunshine and abnormally low amounts of temperature and rainfall. Few showed low sunshine or high temperature and rainfall. The amount of snowfall seems not to bear any particular relation to the incidence of pneumonia.

It is reasonable to assume that the incidence of pneumonia in the Children's Hospital is a fairly accurate index of its prevalence in the community. If this is true, it may be concluded that abnormally warm, rainy weather with little sunshine favors its occurrence, and that abnormally cold, sunny, dry weather is unfavorable to it.

GENGENBACH, Denver.

EARLY CHANGES OF THE LIVER IN ACUTE YELLOW ATROPHY WITH REPORT OF A CASE IN AN INFANT. B. ROMAN and D. SHERMAN, Buffalo Gen. Hosp. Bull. **6**:30 (July) 1928.

The condition developed in a child 8½ months old. The infant was breast fed, and there was no possible cause found in the history. The death occurred in five days following jaundice and vomiting, with acholic stools and bile stained urine. At autopsy, the liver showed a high degree of fatty degeneration, with inflammatory infiltration of the portal spaces, increase of the bile ducts and attempts at cell division. The kidney also showed an extreme grade of fatty change.

SANFORD, Chicago.

FRÖHLICH TYPE OF DYSPITUITARISM WITH EXTREME GENITAL DYSTROPHY. C. WORSTER DROUGHT and B. G. CROWHURST ARCHER, Proc. Roy. Soc. Med. **21**:772 (March) 1928.

A child, aged 8 years, weighed 123 pounds (55.8 Kg.). The blood showed a relative increase in the large lymphocytes. There was extreme dystrophy of the genitalia, the scrotum being entirely absent, the penis small and no evidence of the testicles. The report is accompanied by a full blood count, x-ray reports, measurements and a picture of the patient.

WILLIAMSON, New Orleans.

KUSSMAUL'S DISEASE IN CHILDREN: CASE REPORT. DEBRE, LEROUX, LELONG and GAUTHIER-VILLARS, Arch. de méd. des enf. **31**:325, 1928.

Of the 153 cases of nodular periarteritis thus far reported, practically all are confined to the German literature. The first observation in France is here described, with voluminous clinical and pathologic data. The authors are particularly fortunate in identifying a case of this rare infection in a child, as but eight instances in early life are recorded. A boy of 13 was admitted to the Hospital for Sick Children in Paris, twelve days after the onset of an obscure condition. He appeared mortally ill; profound astenia, intense pain in the limbs and complete anorexia were present; he had a polymorphic cutaneous lesion, a dark red or violaceous eruption, which later became distinctly hemorrhagic and affected the legs, and a similar manifestation over the buccal, lingual and rhinopharyngeal mucosa. On the tongue, on the vault of the palate, on each lip and scattered over the gums were noted vascular, painless nodules about as large as a small cherry

and covered with a grayish exudate. The most prominent digestive symptoms included, in addition to the loss of appetite, continuous vomiting of a bilious character, diarrhea with excessively foul stools, distention and tenderness. The liver was enlarged, and palpation was painful; the spleen was not increased in volume. Urinalysis demonstrated serious involvement of the kidney and the usual symptoms of acute hemorrhagic nephritis. The child sank rapidly after admission and died on the seventh day. Up to this time, Kussmaul's disease had been suspected, but complete differential diagnosis was not made on account of the difficulty of separating it from the syndrome of purpura, of hemorrhagic smallpox or of certain types of leukemia. Autopsy disclosed the typical lesions of periarteritis as described in the original memoir of Kussmaul and Maier in 1866.

AMESSE, Denver.

THE HEREDITARY TRANSMISSION OF THE FILTRABLE VIRUS OF TUBERCULOSIS. FERNAND ARLOING and ANDRE DUFOURT, Presse méd. **36**:753 (June 16) 1928.

Young guinea-pigs were inoculated at birth with the filtrable virus of tuberculosis. While some of the animals did not show any effects, in others a marked retardation of growth occurred. Many of the latter group died of cachexia at from 1 to 2 months of age without true tuberculous lesions but with tubercle bacilli in their lymph glands. The authors therefore advocate the following clinical grouping of new-born infants who inherit the filtrable virus: (1) Those who present the syndrome of athrepsia which sometimes terminates in death; (2) those who show abnormal growth; this retardation is due to an organic intoxication caused either by the presence of the attenuated ultravirus or of the bacilli; (3) those who have inherited a filtrable virus which remains entirely latent and does not produce a disturbance in development. These new facts may cause a complete revision of the subject of the hereditary transmission of tuberculosis.

SANFORD, Chicago.

THE ALIMENTARY ANEMIAS OF INFANCY: THEIR DIETETIC TREATMENT. MAMERTO ACUÑA and PERLINA WINOCUR, Arch. latino am. de pediat. **22**:61, 1928.

The authors discuss anemias of strictly alimentary origin occurring in infants of the first and second period of infancy. There are two types, one, the anemia ferripriva or oligosideremia, described by French and Italian authors. The other type is more complete, with more profound involvement of the hematopoietic system. There is marked diminution of the red cells, with appearance of nucleated cells and enlargement of the spleen.

In the feeding of the infants, milk, cereals and soups predominate. The authors believe that the anemias of infancy, particularly those of the nursing period, are generally due to a prolonged insufficient and one-sided alimentary regimen. Individual idiosyncrasies may vary the picture of the anemias. There may be defects in the hematopoietic system. There may be congenital or acquired diminution or acquired diminution or impairment of vitamin reserves.

The authors believe that there is a subtle connection between these anemias and certain obscure or latent forms of scurvy. For the treatment of the condition, the authors recommend complete change of alimentary regimen and stress particularly a diet of fresh meat, liver, green leafy vegetables and fresh fruits.

SCHLUTZ, Minneapolis.

THE CEREBRAL CONDITION IN BRONCHOPNEUMONIA. C. DELANGE, Jahrb. f. Kinderh. **119**:100 (Feb.) 1928.

DeLange reports a child, aged 3½ months, who became violently ill with spasms, thin stools and vomiting. Marked hypertony was noted, the legs being drawn up toward the body. The cardiac tones were dull and were associated with sibilant râles of both lungs. In spite of the administration of salt solution and

metrazol, the child died on the morning after it was admitted to the hospital. Nine hours later, autopsy was performed. Bronchopneumonia of the left inferior lobe, with edema and purulent bronchitis of the inferior and superior lobes, was discovered. In the right side, the pneumonia affected all three lobes.

The brain showed slight hyperemia in the lower portion. Sections did not show inflammatory processes. The cell content in the pia-arachnoid was increased in places. The upper layer of the cerebral cortex exhibited slight edema.

The corpus striatum was remarkably poor in cells. In the nucleus caudatus, a slight increase in marginal glia was noted. Moreover, the glia were fairly intact in the striatum. In the putamen, the protoplasmal fimbria of the glia cells had spread and revealed a small number of karyokinetic figures.

It seems quite probable that the intense hypertony and tremors were connected with the changes in the striatum, which is an inhibiting center for the corticospinal tract. It is assumed that the decrease in cells was caused by toxic action, as the small ganglion cells are especially susceptible to toxic influences.

THE USE OF CONCENTRATED SOLUTIONS OF METHENAMINE IN THE TREATMENT OF STREPTOCOCCAL INFECTIONS OF CHILDHOOD, PARTICULARLY ENCEPHALITIS AND CHOREA MINOR. B. F. SCHOGAN, Jahrb. f. Kinderh. **119**:210 (March) 1928.

After a brief discussion of colloid chemistry and a history of the introduction of methenamine into therapeutics, Schogan states that he studied the effect of a 40 per cent solution of methenamine in the management of various septic infections of childhood.

The solution is employed intravenously, intramuscularly and intraspinally. The bactericidal effect is due to the liberation of formaldehyde in the body. The high osmotic tension of the concentrated solution stimulates osmotic interchange between various tissues, with the direction of exchange toward the blood stream.

There is an increase in the platelets of the circulating blood, increased rapidity of coagulation and changes in the leukocyte formula due to stimulation of the reticulo-endothelial system.

It is suggested that the concentrated methenamine solution be used in various infections and septic processes such as chronic sepsis, polyarthritis, peri-endocarditis, encephalitis, chorea and cerebrospinal meningitis.

AUTHOR F. ABT, Chicago.

DIFFERENTIAL DIAGNOSIS OF RICKETS AND OSTEOPSATHYROSIS. HANS MEYER, Ztschr. f. Kinderh. **44**:530 (Nov.) 1927.

Meyer reports a case of rickets in a girl, aged 4¾ years. The history, x-ray examination, blood phosphorus and course of the disease showed it to be true rickets. The multiple fracture which she had may have warranted a diagnosis of a combination of rickets and osteopsathyrosis, but the fractures healed synchronously with the healing of the rickets and without new fractures occurring. The healing was so markedly parallel with the rise of the phosphorus curve and the blood serum that one could not avoid the conclusion that the girl had one disease and not two. Typical rickets does not show true fractures; osteopsathyrosis shows true fractures but occurs almost exclusively in children with rickets. It seems, then, that one is dealing here with a rare and special kind of rickets.

EMERSON, Boston.

REPEATED, SEVERE HEMORRHAGES FROM THE NOSE AS A SYMPTOM OF HEREDITARY DILATATION OF THE END BLOOD VESSELS OF MUCOUS MEMBRANES AND SKIN (OSLER'S DISEASE). P. H. J. VAN GILSE and C. POSTMA, Nederl. Tijdschr. v. Geneesk. **72**:2648, 1928.

In this article, the authors describe four persons from two Dutch families suffering from the rare disease described by Osler in 1901. The symptoms consist of the occurrence of severe, repeated epistaxis which is the symptom of hereditary

telangiectasia present in the skin and mucous membranes. In the cases described, only bleeding from the telangiectatic areas of the nose and mouth were observed. The disease is hereditary, following the dominant type, but is not bound to sex. The families observed came from different regions of Holland. In the English literature, there is a description of this disease in a family that also originated from Holland (Ballantype, 1908). The treatment can be only symptomatic.

STOKES-ADAMS SYNDROME IN A CHILD WITH CONGENITAL HEART FAILURE. A. K. J. DE HAAS, Nederl. Tijdschr. v. Geneesk. **72**:3368, 1928.

Haas presents a report of a boy, aged 9 years, who had congenital heart failure of which he had not complained. During the year before the first attack, while performing gymnastic exercises, he suddenly fell down and lost consciousness for a short time. In falling he did not have involuntary urination or bite his tongue. He had never had such an attack before. He complained of headaches, immediately after this attack, felt ill and vomited. Examination showed that the rhythm of the boy's heart was abnormal. A delayed atrioventricular conduction and an extinction of the impulse of propagation in the sinus node were found. As a consequence, long periods of delay in the heart-beat arose, which as far as the rhythm of the ventricles is concerned, are interrupted by autonomic stimulation. In the clinic in which heart block is studied, it has been learned that if in the passing of a partial into a total heart block the autonomic ventricular centrum comes into action for the first time, this occurs with some delay. In the author's opinion, such a praeautomatic pause existed in the beginning of the boy's disease and then caused anemia of the brain with loss of consciousness. After two months, the heart rhythm became more regular and quicker. Graphic investigation of the heart showed that this improvement was not due to a better function of the autonomic ventricular centrum, but to the appearance of a regular atrium rhythm of high frequency, e. g., the so-called "flutter." The boy was treated for a long time with small doses of digitalis.

S. VAN CREVELD, Amsterdam.

TETANY AND ACIDOSIS. PAUL DRUCKER, Acta Paediat. **7**:350 (Feb. 28) 1928.

Drucker presents a rebuttal of the subject.

McINTOSH, Baltimore.

Nervous Diseases

ACUTE TOXIC ENCEPHALITIS IN CHILDREN. R. E. GRINKER and T. T. STONE, Arch. Neurol. & Psychiat. **20**:244 (Aug.) 1928.

In a study of thirteen cases of encephalitis in children suffering with acute infections of varying kinds, including those of the upper respiratory tract, acute otitis media, acute mastoiditis, pneumonia, scarlet fever and septicemia, severe neurologic symptoms were observed clinically, and an acute toxic encephalitis was observed on histologic examination. A fairly uniform clinical course was followed: rapidly developing symptoms of diffuse cerebral involvement, often associated with meningeal symptoms; early stupor; hyperpyrexia and death in from three to four days. The spinal fluid was entirely normal. Two patients recovered. Pathologically, evidence of actual invasion of the brain by microbes was not found. The stress of the toxic agents was directed against the ganglion cells and the vascular system. It is suggested that severe neurologic sequelae may develop from acute toxic encephalitis associated with acute infections if recovery occurs, and that more attention should be directed to encephalitis as an etiologic factor in these sequelae.

SANFORD, Chicago.

SOME ASPECTS OF THE PROBLEM OF SPASMOPHILIA. K. WEJDLING, Acta Paediat. **7**:295 (April) 1928.

Wejdling discusses the chemistry of tetany on the basis of several gratuitous assumptions which lack experimental confirmation.

McINTOSH, Baltimore.

SEROUS MENINGITIS IN INFANTS. Y. F. DOMBROVSKAYA, Pediatriya **11**:105, 1927.

In many infectious diseases of children, especially in infants, an irritation of the meninges, which resembles cerebrospinal or tuberculous meningitis, is observed. Sometimes this irritation of the meninges is so strong that the original disease is not so evident, but the cerebrospinal fluid is clear or only slightly opalescent (cloudy), and contains a normal amount of albumin or an amount slightly above normal, few lymphocytes and little if any bacteria. This meningeal irritation in infants occurs mostly in those with pneumonia, otitis and influenza, but it has been described also in other epidemics, such as dysentery, measles, scarlet fever and chickenpox. Quincke described a type of meningitis which he named serous meningitis, which appears as a secondary manifestation in infectious diseases and which results in irritation and inflammation of the meninges with an increase of the amount of the cerebrospinal fluid, but without any change in the content.

The cases of serous meningitis observed by the author from 1924 to 1926 all occurred in children under 1 year of age. All cases of meningitis were a complication of influenza with otitis, bronchopneumonia and bronchitis.

Conclusions.—1. All cases of meningitis except one were a secondary manifestation of influenza.

2. The clinical form does not depend on the severity of the primary infection.

3. The duration of the meningeal process was between five and twenty-five days and did not depend on the original infection.

4. The constant meningeal symptoms are the rigidity of the muscles at the back of the neck and distention of the fontanels.

5. The cerebrospinal fluid in all cases except one, in which the fluid was opalescent, was transparent with little increase in the amount of fluid and in some cases increase of the albumin; the sediment contained only a few lymphocytes, and the pressure of the cerebrospinal fluid was variable, sometimes not increased.

6. Anomalies in constitution were observed: in six cases, exudative diathesis; in two, neuropathy and in three, spasmophilia; the meningeal symptoms were more serious in cases of spasmophilia.

7. The outcome of the reported cases should be considered as favorable: of twelve patients, eight completely recovered, two had hydrocephalus and two died.

8. Frequently repeated lumbar punctures greatly improve the condition of the patient.

9. Certain of the patients with tuberculous meningitis who were cured probably had serous meningitis of long duration, because of the similarity of the clinical manifestations and the character of the cerebrospinal fluid.

10. In all the cases of secondary serous meningitis described, the primary disease was influenza with its complications, such as otitis and bronchopneumonia; it is probable that the infection from the primary localization in the nasopharynx was carried through the vascular system into the central nervous system.

11. The characteristic symptoms in serous meningitis in children under 1 year are frequent convulsions, absence of constipation (often dyspepsia of infectious character), laryngospasm and tension of the fontanels.

SEVERAC, Philadelphia.

Diseases of the Ear, Nose and Throat

INFANTILE NASOPHARYNX AND ITS CONSTITUTIONAL INFERIORITY. KARL BERNFELD, Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:516 (Feb. 28) 1928.

Testut-Jacob described three types of infantile nasopharynx: the pointed arch, the rounded arch and the low arch. Bernfeld comes to the following conclusions:

1. The normal spacious epipharynx develops from the low infantile type.

2. If the growth of the epipharynx is retarded, an infantile type persists. This constitutes a constitutional inferiority.

3. The infantilism of the epipharynx manifests itself by the frequency of oto-nasopharyngeal inflammations, and by faulty ventilation of the upper respiratory tract. The proximity of the pathologically changed mucous membrane is of importance in regard to the duration and extent of diseases.

4. The constitutional inferiority of the persisting infantile nasopharynx is connected with other constitutionally inferior organs, for instance, with an infantile

Occurrence of Diseases in Persons with Normal Spacious or Infantile Epipharynges

Diseases	Normal Spacious Epipharynx, 60 Treated Patients	Infantile Epipharynx, 60 Treated Patients
Nasopharyngitis	22	40
Hypertrophied tonsils	12	11
Adenoids	18	32
Recurrence of adenoids	3	8
Catarrhal otitis without adenoids	4	19
Catarrhal otitis with adenoids	12	28
Otitis suppurativa chronica mesotympanica nonspecifica	8	13

tube and a shallow tympanic cavity. Hereby the pneumatization of the temporal bone is influenced. Explanation of the frequency of this occurrence requires further anatomic investigations.

5. Digital exploration of the epipharynx reveals conditions in the three dimensions, namely, in the palatosphenoidal, the pharyngonasal and the tubotubal diameters. The palatosphenoidal diameter is shortened most frequently. Investigations at necropsies are desired to further the study of these observations.

AMBERG, Detroit.

Diseases of the Skin

SKIN REACTIONS IN EXUDATIVE DIATHESIS. A. S. ROSENTHAL, Pediatriya **11**:115, 1927.

The skin manifestations in exudative diathesis in the form of eczema or strophulus, or intertrigo in infants, have been known for a long time, but the etiology is not yet established, except for the opinion that it is an autointoxication or a bacterial infection, or lack of internal secretion. Lately, many American and French authors consider the manifestations in exudative diathesis as an increased sensitiveness toward different proteins which are found in the mother's or the child's food. The author used as antigens cow's milk, mother's milk and yolk and white of egg; since these products contain fat and sugar, the author used lactose in a 10 per cent solution and cow's butter as a 10 per cent solution in ether. The reaction was applied to forty-four children from 3 months to 3 years old. Forty-three of them had skin manifestations which are characteristic of exudative diathesis.

Of forty-four children, 50 per cent gave a positive reaction with one of the proteins. The stronger reaction was with white of egg. The author came to the following conclusions:

1. The high sensitiveness to certain proteins was shown only by children with skin manifestations, characteristic in exudative diathesis.

2. In other skin manifestations, the reaction was negative.

3. Of the forty-four cases, 50 per cent reacted toward some of the proteins.

4. Among the positive cases the reaction occurred more often with yolk and white of egg.

5. The intensity of the reaction varies, and the size of the surface of reaction varies from 2 to 6 cm.

6. A strong parallelism exists between the strength of the reaction and eosinophilia.

7. With the improvement of the eczema, the reaction is less strong and the percentage of the eosinophils in the blood diminishes.

8. The reaction has nothing to do with the age of the children and the way they are nourished.

9. If further observation will prove the high sensitiveness of the skin to different antigens, the latter could be used for treatment (desensibilization).

SEVERAC, Philadelphia.

Surgery and Orthopedics

PSEUDO-APPENDICITIS IN CHILDREN. FRANK SHAPIRO, M. J. & Rec. **127**:383 (April 4) 1928.

Acute appendicitis in children is sometimes difficult to diagnose and in most cases should be diagnosed more by exclusion of other diseases than by positive physical symptoms.

Appendicitis in children should be differentiated from the following diseases: gastro-intestinal disturbances; acute intestinal obstruction; intussusception; cystitis; pyelitis; acute nasopharyngeal infection; influenza; typhoid fever, and pneumonia. In suspected appendicitis, one should make a thorough physical examination, having all possibilities in mind.

WILLIAMSON, New Orleans.

SURGICAL TREATMENT FOR PYLOROSPASM IN INFANTS. H. VON WILLEBRAND, Acta Paediat. **7**:27 (April) 1928.

Von Willebrand cites one poor result following medical treatment and two instances of excellent surgical results as arguments in favor of the Rammstedt operation.

TREATMENT FOR TUBERCULOSIS OF THE BONES AND JOINTS. R. KYRKLUND, Acta Paediat. **7**:173 (April) 1928.

Kyrklund followed the practice of Kirsch in treating tuberculosis of the bone with direct exposure to sunlight and to therapeutic lamps, with the application, when possible, of Bier's hyperemia, by the use of the least immobolizing apparatus consistent with the patients' comfort, and by the internal administration of sodium iodide. He treated 157 patients in this way over a period of five years. The average period of observation was from two to three years. In spondylitis the mortality approximated 22 per cent, while 50 per cent of the patients were discharged in good condition and did not suffer a relapse; the average duration of treatment in the successful cases was 15.9 months. Kyrklund had good results with the use of graduated cushions applied beneath the gibbus, and was able to dispense with plaster casts and frames.

In the group of patients with tuberculous hips, the mortality was 6 per cent, while 31 per cent were still under treatment and 62 per cent were discharged cured, with no instance of relapse. Treatment in the latter group lasted, on the average, 10.9 months.

Of the children with involvement of the knee joint, 67 per cent were cured, 40 per cent with a residual mobility of the joint of 90 degrees or more. Here the average duration of treatment was 19.8 months.

The results show considerable improvement over those previously reported from Finland, and the author attributes this to his therapeutic regimen.

INTESTINAL TUMORS IN INFANCY. A. JOHANNESSEN, Acta Paediat. **7**:213 (April) 1928.

Johannessen reports a case of sarcoma of the rectum in an infant of 1 year.

MCINTOSH, Baltimore.

INTRACARDIAL INJECTION OF EPINEPHRINE DURING COLLAPSE CAUSED BY CHLORETHYL-NARCOSIS. G. JOHNSON, Norsk. Mag. f. Laegevidensk. **89**:478, 1928.

A sudden collapse occurred in a girl, aged 3 years, during ethyl chloride anesthesia preceding a tonsillectomy. When the narcosis had lasted for two minutes and one tonsil had been removed, the child became suddenly pale and pulseless and was moribund. Artificial respiration did not have any effect. As a last resort, 0.5 cc. of epinephrine was injected intracardially, with the result that the heart began to beat within half a minute. Later, the respiration returned. The other tonsil was removed quickly, and the child recovered after one hour.

TOVERUD, Oslo.

Miscellaneous

THE EFFECT OF CARBON ARC IRRADIATION ON THE HEALTH OF A GROUP OF INFANTS. LOUIS H. BARENBERG and J. MELVIN LEWIS, J. A. M. A. **90**:504 (Feb. 18) 1928.

Systematic exposures to the radiations of the carbon arc lamp did not reduce the incidence of respiratory infections in a series of infants treated for three months. During the first six weeks, growth in weight and height was greater in the irradiated group than in the nonirradiated group. In the second six weeks, the opposite was the case. In spite of repeated exposures to the rays of the carbon arc lamp, the pigmentation of the skin was slight, as compared with the tanning resulting from a few days' exposure to sunlight.

INTRAPERITONEAL USE OF DEXTROSE IN THE TREATMENT OF DISEASES OF CHILDREN. HEYWORTH N. SANFORD and P. L. HEILMEYER, J. A. M. A. **90**:737 (March 10) 1928.

The authors have found that a 5 per cent solution of dextrose may be given intraperitoneally without reaction of any kind, if the dextrose is prepared by dry sterilization. The use of this solution may be extended to some acute infections and to postoperative conditions as well as to nutritional disturbances. To be effective, the dextrose should be given in 100 cc. quantities, repeated frequently.

ACIDOSIS IN METHYL SALICYLATE POISONING. J. G. M. OLMSTED and C. A. ALDRICH, J. A. M. A. **90**:1438 (May 5) 1928.

A pronounced acidosis was noticed in two children after ingestion of oil of wintergreen. In most of the other cases reported, symptoms of acidosis were prominent. A summary of the results of treatment lends credence to the idea that acidosis is the dangerous pathologic lesion. Early alkalinization in the treatment of these two children resulted in overnight cure.

BONAR, Salt Lake City.

THE OCCUPATIONAL DISORDERS OF THE NEW YORK PUBLIC SCHOOL CHILD. WILLIAM ROSENSON, N. Y. State J. Med. **28**:313 (March 15) 1928.

In this paper read before the Medical Society of the County of New York, the author severely criticizes the New York City Public School system in the care of the pupils, numbering more than one million, under its supervision.

The recess for lunch is now only fifty minutes. The necessary rush for this meal produces much malnutrition and nervousness. The school lunches are unsatisfactory. So much stress is put on perfect attendance that the children conceal symptoms in order to avoid criticism. Such children may easily injure themselves and prove a source of infection to others. Children should not be kept after school for punishment. The law requiring 200 minutes a week for physical training and games, and the requirements for education in hygiene have sometimes been met by adding two hours to one day each week. There is no definite regulation regarding

the amount of home work. The lavatories are frequently insanitary and are not protected from intruders. Children from 6 to 7 years of age frequently have to begin school at 8:10 a. m. because of the double session. In some schools with double session, the children are not provided with closets for their clothing, and many wear their wraps during school hours.

The constant rush and long hours have a serious effect on many children. Classes sometimes run as high as fifty to a teacher. It is recommended that a commission of physicians be established to pass on all matters concerning children's health in public school.

This severe indictment of the school systems was discussed by Louis I. Harris, Commissioner of the Department of Health in New York City. He agreed with Dr. Rosenson, and stated that the superintendent of school and other leaders in the educational system of the city see clearly and fully the disadvantages of the present system. "We must have an enlightened and popular sentiment to support a radical change in the present methods."

AIKMAN, Rochester, N. Y.

REMARKS CONCERNING THE USE OF ARTIFICIAL LIGHT, ESPECIALLY IN TUBERCULOSIS. AXEL REYN, Phys. Therap. **45**:559 (Dec.) 1927.

The author, director of the Finsen Institute, states that for local light treatment of tuberculosis of the skin the concentrated carbon arc light should always be used. This light penetrates much more deeply than the rays of the Kromayer mercury quartz lamp which produces chiefly the less penetrating shorter ultraviolet rays of less than 3,600 angström units wave length. Lupus vulgaris, being a deep-seated process, requires the more penetrating long wave length rays of from about 4,000 to 4,300 angström unit lengths. The latter rays are produced in abundance by the Finsen carbon arc lamp. For general light baths, used in conjunction with local treatment and especially in surgical tuberculosis, the author believes the carbon arc lamp is far superior to the mercury lamp because the radiation of the former more closely approaches sunlight in its physical and chemical properties. In cases of lumpus vulgaris of not more than four years' standing when treatment was instituted, 70 per cent of cures were reported for persons treated with local light applications alone and 90 per cent in the group receiving both local light treatment and general light baths. Reyn does not consider the production of erythema or pigmentation necessary for a successful outcome of treatment, but stresses the value of applying the entire light spectrum rather than a narrow band such as an exclusively ultraviolet radiation.

H. R. FOERSTER.

POVERTY, NUTRITION AND GROWTH: STUDIES OF CHILDREN IN CITIES AND RURAL DISTRICTS IN SCOTLAND. Medical Research Council — Special Report Series No. 101. D. Noel Paton and Leonard Lindlay, His Majesty's Stationery Office, London, 1926.

Although the importance of child welfare is widely recognized for "the character of the adult population of any country, its health, vigor and working capacity is determined by the development and growth of the children. As this is in turn influenced not only by environment after birth, but also by factors working while the child is yet unborn," little is known accurately of the factors that make for normal development and growth of the child. It has been shown that the children of the poor of the large cities have a higher infant mortality and that those who survive are of poorer physique than those of the same social class in rural communities, but although many hypotheses — poverty, bad housing, defective feeding, lack of fresh air, inefficient material care, heredity, and other conditions — have been advanced in explanation, conclusive evidence, based on proper investigation, has not been advanced to support them.

This monograph reports the results of an investigation undertaken in Scotland by a number of workers in collaboration with the authors, to determine more precisely what conditions modify the growth and nutrition of the child. One hundred children of each age and sex from the slums of Glasgow, Edinburgh, and Dundee were studied and compared with a similar number in a Scotch agricultural district and in a rural coal mining area. The children were grouped into three phases of life: (1) from birth to 1 year; (2) from 1 to 5 years; (3) from 6 to 14 years; emphasis was laid on the second group — the preschool period — because it is this period that should yield the most valuable information as to the effects of conditions in the home. It was found that up to the age of 5½ years, the children in agricultural districts are the tallest and heaviest and those in districts of the slums are the lightest and shortest. There seems to be a lag in the average increase in height and weight of children of the urban districts between the ages of 6 and 18 months, and this is more noticeable among girls than boys. After eighteen months the increase in weight in boys is as rapid in urban as in rural districts, and faster among girls of the urban districts. In the same period the increase in height is quicker in the urban than the rural communities. During the first eighteen months, girls of the rural districts increase in height and weight more rapidly than boys; in the cities the reverse is true. It was impossible to correlate any environmental factors with the lag in growth in the first eighteen months of the life of the slum child, but "whether environmental conditions are or are not responsible, it seems manifest that after that age the inherited growth impulse is sufficiently potent to carry it so completely on to the average size of its parents, that the influence of environment is not indicated on the curves of growth of the average of the whole number of children, and it is also evident that the average diet of the population must be at least sufficient to supply the material and energy required for growth."

A large number of the factors that were thought to influence the growth and nutrition of the child of the slums were investigated. It was found that the size of the family, health of the mother during pregnancy, the general health of the mother, the mother's occupation and the size of the income did not have any effect on the growth of the child. There was small correlation between the total income and the calories of the diet, but the energy value of the diet depended more on the marketing than on the total income. Inadequate diets were not restricted to the low income groups, for even in families with larger incomes there existed a considerable degree of inadequacy. The relation between the diet and the growth of the children in twelve typical urban families was studied intensively. It was found that the average value of the diets was very low — 2,119 calories per person a day. There was a certain but not close correspondence between the income and energy values of the diets. The children averaged 9.1 per cent in weight and 4.8 per cent in height below the generally accepted standards. The children of families with incomes below the mean fell short of the standard in weight by 10.8 per cent and in height by 5.8 per cent, while those of families with incomes above the mean fell short by 5.5 per cent in weight and 2.5 per cent in height. When arranged in families spending more than the mean and those spending less on food, the difference was less marked; in the former the weight was 7.5 per cent below, the height 3.8 per cent; in the latter the weight was 10.3 per cent below and the height 5.5 per cent below. When the group was divided into families with a diet above the mean of calories and those below, it was found that the children of the former averaged 12 per cent in weight and 5.5 per cent in height below the standard, while of the latter were in weight 6.2 per cent and in height 4.0 per cent below. One family was studied three times in three years. The average caloric value of the diet was lower than the average of the families studied in the city in which they lived. The rate of growth of the children, however, corresponded to the mean growth of all the other children. The height and weight

of the older children showed a tendency to be below the mean although that of the younger children approximated closely to it.

The results obtained from these studies were contrasted with similar studies on rural families of a similar social status, and the results indicated that even on the low intake of calories and a milk supply of about 0.4 pint a day, the child of the city slums after 18 months of age grows at the same rate as the country child who has a much greater number of calories. Age for age, the country child from 1 to 5 years was from 10 to 11 per cent heavier than the child of the slums. It would seem that the usually accepted number of 3,000 calories as the minimum requirement per person a day is excessive, and that from 2,500 to 2,700 is nearer the correct figure. The comparatively high correlation between calories in the diet and the income, and the absence of any correlation between the weights of the children and the income seemed to indicate that diet is not the all important factor in determining growth that it is supposed to be. The small size and weight of the child of the slums may be more intimately related to heredity than to diet.

Breast feeding is supposed to exert a favorable influence on the future growth of the child. It was found that the majority of mothers nurse their children during the first month and if they do not discontinue then, usually continue till from the sixth to the ninth. There seemed a correlation between the weight of the child up to the ninth month and the duration of breast feeding, but after this age no sensible relationship was demonstrable. Breast feeding did not seem to have any effect on height. After the period of weaning, the health of a child who had not been breast fed at all was as good as that of the child who has been breast fed the whole time. This might be accounted for by the higher death rate among artificially fed infants with a consequent survival of the fittest, so that the healthiest of the artificially fed were compared with the average of the breast fed; there might be some retardation in growth from the change of feeding which would have caused recovery sooner in those that were weaned earlier.

Over-crowding and lessened air space per person did not seem to have any influence on the growth of the child under 1 year of age. Between the ages of 1 and 5 it had some influence, and during the school period it resulted in a smaller and a lighter child. The health of the mother did not appear to be affected by over-crowding, poverty or the size of the family. The efficiency of the mother was the factor most definitely related to the growth and nutrition of the child, and was related to material health, over-crowding, size of family, and possibly to the mental capacity, but not to income. Maternal efficiency, however, only operated as a factor during the first two years of life.

The age of the mother had a slight correlation to the height and weight of the child. Infants of young and old mothers were inferior in height and weight to those of mothers of medium age; this was noted particularly throughout the first half of the first year of life. By the last three months of the first year, the inferiority of the children of young mothers had disappeared, but that of the children of older mothers remained. The order of birth had a greater influence. Infants of the first and over the eighth pregnancy were lighter and shorter than those of the intervening ones. The inferiority of the first born was replaced at the end of the first year by a definite superiority, but that of the last born of families over eight was still apparent at the end of the year. Paternal habits were not investigated sufficiently intensively to lead to any conclusions.

Somewhat similar results were obtained from the study of agricultural families and those of rural miners. In both groups it was found that neither income, air space per person, the size of the family, nor the health of the mother had any effect on the growth or nutrition of the child. The health of the mother was dependent on the size of the family and in the former group seemed correlated with the income, but not in the latter group. Maternal efficiency seems related to the income and the size of the family.

No definite factor seems to have been discovered to account for the lag in growth and nutrition of the child of the city before the middle of the second year of life. Future investigations should concentrate on this period.

PEARSON, Philadelphia.

A STOOL TO FACILITATE X-RAY EXAMINATIONS OF INFANTS. GEORG ABRAHAM, Monatschr. f. Kinderh. **39**:100, 1928.

Abraham describes the apparatus.

GERSTLEY, Chicago.

RESULT OF AN INQUIRY INTO THE MANNER IN WHICH THE CHILDREN OF THE PRIMARY SCHOOL OF WARSAW PASS THEIR VACATIONS. S. PIOTROWSKI and W. PIOTROWSKI, Opieka Nad Dzieckiem (Polish) **6**:95, 1928.

In this inquiry, the principal questions asked were: What is the frequency of sojourns in the country for vacation? What is the duration of these? What number of children quit the city at the expense of their parents and what do the children passing their vacations in the city do with their time? Questionnaires were distributed to children aged from 8 to 15 years. The following results were obtained. From the 9,300 responses received, it was found that 75.3 per cent of the total number of children have made sojourns in the country and that 24.6 per cent have never been there. Among the children who have been in the country, 6.3 per cent live or have lived about the city; 24.16 per cent make an annual trip to the country and 44.3 per cent make only occasional trips. Replies for the year 1926 showed that 55.4 per cent had left the city and that 45.6 per cent had passed their vacations in town. Considering a sojourn in the country of at least six weeks as a necessary minimum for the health of the child, the authorities have established by means of this inquiry that 35 per cent of those children who left the city had a sojourn of too short duration.

The inquiry established the solidity of the connection between relatives inhabiting the country and the city. Most children leaving the city for the country stayed with relatives free of charge. The minority paid for their vacation. Sixty-nine per cent of the vacations were free and 31 per cent were paid for.

The children who passed their vacations in the city played about alone in the courts, streets, suburbs, and on the banks of the Vistula, being taken by their parents not oftener than once a week to the parks and public gardens. If one figures from the percentages established and the total number of children of the same age and social strata, 42,700 children have not had a normal vacation.

Comparing this number to the funds provided by various agencies and by the city, one finds that the means are altogether insufficient for remedying this condition.

The authors urge the introduction of a special tax for the children paid by those who are privileged to leave the city in the summer. It should be general, obligatory and progressive. It is the duty of the city to establish it.

AUTHOR'S SUMMARY.

CARE OF WORKING BOYS AND GIRLS. ADOLF KLESK, Opieka Nad Dzieckiem (Polish) **6**:130, 1928.

To meet the need of working children in their free time between work and evening schools, clubs should be provided where they may gather for lectures or conferences and where they may have a bath or be examined by a doctor in case of sickness or excessive fatigue. Otherwise the evil of the street has a bad influence. To this same group belong children of the common schools who sell matches, flowers, toys, etc., in the cafés at night.

The record in Polish cities during the Austrian occupation shows that one half of all school children of school age had to work for their living, and in many places

had to work in their homes. Often the period of work was from six to ten hours a day. For these reasons, child labor before the age of 10 or 12 years should be absolutely forbidden; this should be especially forbidden when the hours are from 8 p. m. to 8 a. m.

The children working regularly should be enrolled in either official employment offices or workmen's beneficial associations, where they could be examined by the doctor. In factories, children of 12 years of age and over are allowed to work only during the day. During the school year, they cannot work longer than three hours a day. This labor should not interfere with the schooling of the child. The schools should keep an exact list of children who are employed and how long they are required to work.

AUTHOR'S SUMMARY.

Society Transactions

CHICAGO PEDIATRIC SOCIETY

Clinical Meeting, April 17, 1928

MAURICE BLATT, M.D., *President, Presiding*

PYELITIS (ONE KIDNEY). DR. JOSEPH K. CALVIN.

The left kidney of the patient, a girl, aged 5 years, had been removed when she was 6 months old, because of an abscess and some congenital anomaly of the left ureter, after the right kidney and ureter had been pronounced normal. The child developed normally until she was about 4 years and 3 months old when, one week after an attack of chickenpox, she developed symptoms of pyelitis (frequency and dysuria), with a temperature ranging from 101 to 105 F. Repeated examinations of the urine did not reveal pus until the fifth day of fever, when a large amount was found. After a few weeks of septic temperature and pyuria, the temperature returned to normal and remained there for about six weeks. The pus disappeared from the urine. Then there was a recurrence of fever, with pus in the urine, which persisted for three or four weeks. There were three such recurrences, the longest time the urine remained free from pus after the onset of fever being five days. A roentgenogram of the kidney and bladder did not show stones. Cystoscopy was performed, but no ureteral catheterization was attempted for fear of causing complete suppression of urine. There was no evidence of tuberculosis in the bladder; the Mantoux test and guinea-pig inoculation likewise gave negative reactions for tuberculosis. The unusual and interesting feature of the case was the fact that the urine remained free from pus for from three to five days after the onset of fever, although there was but one kidney. It might be that a valve or diverticulum in the pelvis of the kidney held back the pus but allowed the urine to escape until something happened to break down this resistance. The use of methenamine and hexylresorcinol failed to clear up the condition permanently.

TUBERCULOSIS OF THE HIP. DR. FREDERICK C. TEST.

CASE 1.—A girl, about 9 years of age, began to develop "night cries," two years before presentation. She apparently did not feel discomfort during the day. She did not present any unusual symptoms aside from the night cries until about eight months before presentation, when she began to limp. She has been under the care of an orthopedic surgeon for only a few months. There was a flexion deformity of the left hip amounting to 45 degrees when traction apparatus was applied, with about 45 degrees external rotation and perhaps 20 to 25 degrees of abduction of the leg. There was marked diminution of rotation, only slight abduction and adduction, but there remained about 20 to 30 degrees in flexion and extension. The roentgenograms showed a condition found in only about 20 per cent of cases of hip and joint disease, in which the primary infection is not in the epiphysis, but in the upper part of the acetabulum. These patients recover with a minimum of deformity and maximum of function. Both legs are now under traction; the right leg is held vertically in order to obliterate the compensatory lumbar curve that usually occurs in such cases if the uninvolved leg is left untreated. The angulations are steadily diminishing.

CASE 2.—A girl, aged 4 years, was admitted to the hospital in August, 1927, with tuberculosis of the spine. A history of the duration of the condition was not given. She had a distinct kyphosis in about the midthoracic region, representing almost 90 degrees of angulation. She was placed supine on a Bradford frame,

so arranged that the highest point of the curve came under the kyphosis. She has remained there since that time, and there has been definite improvement. She had a considerable elevation in temperature on admission, and still has a little fever at times. The interesting thing is that, by keeping her in continued hyperextension the kyphosis has been reduced, and she has been much more comfortable and in much better general health than on admission. If she were older, her chance of getting well would be much less than at present. My experience with tuberculosis of the spine in colored persons, in whom the invasion occurs in early adult life, is that it nearly always ends fatally, often very rapidly. The roentgenograms in this case show a destruction of the bodies of the eighth and ninth thoracic vertebrae, which are flattened out so that they are almost wafer thin. This summer, the child's daylight hours will be spent prone on the reversed frame, with her nude body exposed to sunshine as extensively as possible.

NEUROMUSCULAR DISTURBANCES. DR. MAURICE L. BLATT.

A moving picture film was shown of several interesting neuromuscular diseases of childhood. In the first film, a child 10 months old displayed fine fibrillary twitchings. Meningeal irritation in infancy is often evidenced by this type of convulsive seizure. In this case, the left side only was involved. The diagnosis was tuberculous meningitis, resulting from exposure to her father's recognized case of tuberculosis; meningeal irritation showed the unilateral tremor, and indicated an increased pressure of the spinal fluid, by a positive globulin reaction and a cell count of 120 cells per centimeter, apparently all lymphocytes.

The second film showed children from 9 to 12 years of age, who demonstrated various types of chorea. Carious teeth, deciduous and abscessed, were present in most instances. Tonsillar involvement preceding the infection was common in many of these cases. So far, but one satisfactory treatment has been found, namely, prophylaxis. Teeth and tonsils containing foci of infection should be removed immediately. Dr. Blatt said that although he recognized that the removal of deciduous molars has a bad effect on the dental arch, he preferred a poor dental arch to an endocarditis, which occurs in so many of these cases. He controls the disease with large doses of salicylate and sodium bicarbonate, rest in bed, warm baths and if necessary phenobarbital.

Children with latent tetany often have complications of spasmophilia, showing the characteristic carpopedal spasm and the facial reflexes described by Chvostek and shown by the children in this film. The disease is closely related to rickets, which seems to be a factor. Evidence of tetany, however, does not occur unless excited by some mild infection, such as disease of the upper respiratory tract. The characteristic observations are a low plane of irritability to electrical and mechanical stimuli and a diminution of blood calcium to less than 7 mg.

The next film showed a group of children with postencephalitic disorders, to which increasing attention has been given since the influenza epidemic in 1918. The first case was one in which the disease occurred five years ago. The child had some emotional instability and the characteristic fixed facies, and displayed the unusual phenomenon of apnea following intense hyperpnea. Dr. Blatt thought that undoubtedly the corpus striatum was involved. The next case was that of a child, aged 3 years, who showed positive reactions to the Babinski, Oppenheim and Gordon tests, with athetoid movements of the hands and feet, which were thought by neurologists to evidence degeneration of the putamen. In this case, the child watched the floor as she walked even when receiving assistance.

The last film was exceedingly interesting and instructive, demonstrating hydrophobia, which is occurring with greater frequency throughout the world. There are two types in both man and animals: paralysis or dumb rabies and the furious or convulsive type. This 4 year old child came into the hospital one hour before the pictures were taken, with a history of having been bitten on the face, leg and forearm seventeen days previously. The illness began with irritability on the fourteenth day, and the convulsive seizures continued intermittently until death, which occurred fourteen hours after the roentgenogram was taken. The only

known medication for a patient in the throes of this dread disease is a sedative, and this child responded fairly well to large doses of chloral and bromide administered by rectum. He remained conscious through all of his violent convulsive paroxysms, almost to the moment of his death. No antirabic treatment had been administered after the bite.

NIEMANN-PICK'S DISEASE (LIPOID CELL SPLENOHEPATOMEGALY, PICK; LIPOID HISTIOCYTOSIS, BLOOM). DR. JULIUS H. HESS.

This child had a sister who died when 11 months of age after a history similar to that in the present case. Siegmund has also reported an instance in which a second case of a large liver and spleen occurred in the same family. It is interesting that at least ten of the fifteen cases of this disease on record occurred in Jewish children, most of them of Jewish families from south central Europe. Five of the fifteen have been reported from Chicago clinics. The disease is one of early childhood. The child whose case I am reporting died at 15 months of age.

Analysis of Blood and Fat Content of Liver in Case of Niemann-Pick's Disease

	Blood		
Analysis per 100 Cc. of Blood	Our Case (McFate) Mg.	Normal Specimen, Mg.	Per Cent of Increase
Phosphatid phosphorus	24.0	14–14	70
Cholesterol	231.0	140–170	35
Total fatty acids	560.0	290–420	33

	Spleen		Liver	
Analysis in Grams per 100 Grams of Dried Material	Our Case (McFate) per Cent	Normal Specimen (Bloom and Kern) per Cent	Our Case (McFate) per Cent	Normal Specimen (Wahl and Richardson) per Cent
Moisture	78.44	81.4	76.20	.....
Total lipoids	61.29	22.851	61.57	22.99
Fatty acids	2.91	1.039	2.51	.....
Lipoid phosphorus	4.25	1.287	4.14	.....
Lipoid nitrogen	9.70	12.925	12.48	.....
Cholesterol	8.76	2.424	7.37	.....

She was admitted to the hospital twenty-nine days before death. Splenic puncture was performed, and the foam cells were found in the fluid. Enlargement of the spleen and liver was noticed when the child was 6 months old. Some x-ray therapy was administered, and the general condition is said to have improved for a time. The child became worse, however, and was taken from one hospital to another.

Of more than passing interest is the fact that five of the fifteen children on record with this disease, including this child, had marked mongolian spots. Five of the children also showed the cherry red spot and macular degeneration characteristic of amaurotic family idiocy. All of the children showed mental deterioration.

This is the first report of a chemical examination of the blood and, I believe, of the fat content of the liver.

There are several things to remember in making a splenic puncture. The child should be at least partially anesthetized, or sufficiently under an opiate to keep it quiet. We use a 20 gage needle, 3 cm. long, and prefer a needle with a blunt point. This is attached to a 5 cc. Luer syringe. Only a few drops of material from the spleen is necessary for diagnosis. In order to retain the content of the syringe, the plunger should not be allowed to spring back during withdrawal of the needle.

CHRONIC NEPHRITIS, HYPERTENSION, NEUROCHORIORETINITIS. DR. CHARLES K. STULIK, JR.

This girl, aged 10 years, complained of progressive frontal headache of three months' duration and nausea and vomiting of six months' duration. She had had a subicteric pallor for some time, anorexia for several months, double vision and had seen black spots before the eyes. The history did not mention edema or epistaxis. There was nothing unusual about her birth or development. The tonsils had been large and cryptic since early childhood; removal was recommended but not carried out. She had measles at 4 years of age and repeated middle ear infections; there was no history of rheumatism or chorea; her teeth were in good condition. There was, however, a bilateral cervical adenitis. The blood pressure was: 220 systolic and 178 diastolic. The heart was slightly enlarged and showed a moderate mitral lesion. There were multiple petechial hemorrhages, especially over the axilla and about the finger tips. Examination of the eyegrounds showed the disks blurred and multiple hemorrhages. Muco purulent material was found in the left maxillary sinus. The hemoglobin content was 50 per cent; the white cells numbered 10,000 and the red cells 2,280,000.

Follicular tonsillitis developed while the patient was in the hospital, accompanied by a mild otitis media and a rise in temperature varying between 102 and 103 F. The Wassermann reaction was negative.

Chemical examination of the blood on March 6 revealed 37 Gm. of urea nitrogen, 3 Gm. of uric acid, 2 Gm. of creatinine and 214 Gm. of sugar. A month later, urea nitrogen amounts to 30 Gm. and the uric acid and creatinine have remained the same. The urine repeatedly has large amounts of albumin, leukocytes and casts. Under a regimen of rest in bed, citrate and limitation of protein and salt, the blood pressure dropped to 130 systolic and 80 diastolic, and the subjective symptoms disappeared. The prognosis, in spite of the relatively low percentage of creatinine, is poor as to both life and recovery.

EMPYEMA (POST PNEUMONIC). DR. PHILIP ROSENBLOOM.

CASE 1.—A child, aged 11 years, with a pronounced case of dyspnea, had one of the few unusual types of empyema observed last winter. She became ill four weeks before presentation, with pain and a high temperature; she probably had pleurisy. She was admitted to the hospital as having a case of probable pericarditis, possibly because of the absence of heart tones in the region of what should have been the heart. There was complete flatness over the entire left side; examination posteriorly did not show any bronchial breath sounds. Anteriorly on the right side, one could feel the apex beat at about the nipple line. We removed 500 cc. of fluid last night and 600 cc. this morning. With the 1,100 cc. of fluid out of the left chest, the apex is still under the right nipple, but she is much more comfortable. She will be transferred to the surgical department in the morning.

CASE 2.—A child, 10 years old, was taken sick on March 18, with severe pain on the left side of the chest and a cough. She was admitted to the hospital on March 24. She has a history of bronchial breathing at the base of the left lung, with flatness and with what was supposed to be a to-and-fro friction rub over the base of the heart. There was a question as to whether the condition was pericarditis with effusion, but there was neither bronchial breathing on the left side of the chest nor Bamberger's sign. She has marked edema of the left side of the chest. She does not have a pericardial rub now, and she is more comfortable than we would expect in a case of pericarditis. There is flatness throughout the entire left side of the chest, with diminished breath sounds. The heart is pushed out toward the right, probably because of an accumulation of pus. Aspiration posteriorly on several occasions has not shown pus, but on the day before presentation when the needle was inserted, somewhat anteriorly, 60 cc. of pus was obtained. This is probably an encapsulated empyema.

TYPHOID FEVER. DR. A. LEVINSON.

A small colored boy, aged about 5 years, entered the hospital on April 5. He had had a cold for five weeks and a high temperature of rather sudden onset, continuing for five days. On admission, there was dulness in the right base posteriorly, and numerous râles scattered over the chest. The tonsils were congested, and the ear drums were bulging. The temperature varied between 101 and 105 F., although the child did not look acutely ill. A diagnosis of pneumonia of the right lung with bilateral otitis media and tonsillitis was made, although the white cell count of 6,500 did not conform with the diagnosis of pneumonia. A diagnosis of epidemic influenza was considered. On admission the patients' ear drums were incised. A few days later, the drainage having ceased, they were again incised. The right ear began to drain profusely, with a bloody discharge. At this time, a Widal test was made; the reaction was reported positive. Our experience is that the Widal test usually gives positive results in high dilutions. A diagnosis of typhoid fever was considered, but not confirmed, as the spleen was not palpable (and is not palpable even now) and the illness was of sudden rather than gradual onset. The ears continued to drain, the temperature remaining high. The white cell count, however, was only 9,600. A double mastoidectomy was done, but the temperature still remained 103 and 104 F., and the white cell count was 7,200. All this time, the child did not look acutely ill. The Pirquet and Wassermann tests gave negative results. Another Widal test was made and was positive in a dilution of 1 : 1,280. This, I think, is definite evidence that the child has typhoid fever. Stool cultures were negative for some time, but during the last two weeks have been positive. Blood cultures were negative.

This case shows that a diagnosis of typhoid fever should never be excluded, in spite of a history of sudden onset of symptoms, and the absence of splenic enlargement.

PNEUMONIA. DR. F. L. SOUTH.

This girl, aged 7 years, was admitted to the hospital on March 3. She gave a history of having had bronchopneumonia at 1 year of age and whooping cough at the age of 5, complicated by pneumonia. She had an attack of lobar pneumonia in December, 1927; a second attack, in January, 1928, did not result in complete recovery, for she has had a constant high temperature, a cough, has expectorated a large amount of yellowish or greenish-gray foul smelling sputum, and has suffered from occasional attacks of pain in the left portion of the chest.

Physical examination showed that the left wall of the chest is less full and less mobile than the right. On the left wall posteriorly, there is impaired resonance from the apex to the sixth interspace, with flatness, increased vocal and tactile fremitus, and bronchial breathing over the remainder of the lower lobe, with disseminated, coarse, moist and crackling râles. Anteriorly, there is dulness, bronchovesicular breathing and many moist râles. Laterally, there is a marked friction rub, numerous crepitant râles and tenderness on palpation. On the right, the chest is somewhat bulging and resonant, the breath sounds are exaggerated and many moist râles are present, although fewer than on the left side.

The borders of the heart are normal, with no displacement. There is marked clubbing of the fingers.

The temperature on admission was 104 F. It subsided gradually in the course of a few days, to fluctuate daily between 98.6 and 101 F., for a period of about twenty-eight days. Then it again went up to the former high level with the onset of an acute attack of pain in the left part of chest, lasting about three days. There has been abundant expectoration of yellow and tenacious sputum in the morning. Microscopic examination of stained smears has shown no tubercle bacilli. The Pirquet test has given repeated negative results; urinalysis also gives a negative reaction.

X-ray examination on March 5 indicated some type of irregular consolidation, which might well be of a preabscess character. An examination a month later,

coincident with the attack of pleurisy, suggested the presence of a large amount of fluid. Puncture of the chest on three different occasions, however, has been negative for fluid or pus.

A tentative diagnosis has been made of chronic, fibrous, interstitial pneumonitis, with a complicating bronchiectasis, a recent acute fibrinous pleuritis and probable exudate in the pleural cavity, and a compensatory emphysema of the right lung. In the differential diagnosis, we have to consider pulmonary abscess, unresolved pneumonia, chronic fibrinous tuberculosis, atelectasis, congenital bronchiectasis or cyst, and tumor. The treatment has been only of a general hygienic and supportive character, with the administration of potassium iodide internally. Thoracoplasty has been suggested.

MITRAL AND TRICUSPID RHEUMATIC ENDOCARDITIS WITH PULSATING LIVER. DR. SAMUEL J. HOFFMAN.

This boy, aged 12 years, was admitted to the hospital in February, 1927, with a history of dyspnea for seven months, swelling of the legs and abdomen and cough. He had had frequent sore throats, and tonsillectomy was performed four months before presentation. The history did not mention recent acute exanthems, chorea or rheumatism.

At the time of his admittance, examination disclosed a decompensated mitral endocarditis; orthopnea was so pronounced that a paracentesis of the peritoneal cavity was done, and 1,000 cc. of fluid was slowly removed. This fluid was a transudate. Distinct relief followed; compensation subsequently occurred, and he was discharged from the hospital.

On March 14, 1927, he was readmitted with the report of a double mitral lesion and left hydrothorax. He was readmitted again on Feb. 25, 1928, with a report of decompensation that included edema of the face and feet, swelling of the abdomen, vomiting and orthopnea. He was continually afebrile. The cornea revealed many phlyctenules.

Examination at this time revealed matted large glands in the right anterior triangle of the neck. The apex beat was diffuse in the fifth and sixth interspaces at the anterior axillary line. The right border of the heart was 6 cm. to the right of the midsternal line and the left border of the heart was in the left axillary line. A presystolic thrill was palpable over the left side of the precordium. Presystolic and systolic murmurs were heard over the mitral area, and a double murmur was heard over the tricuspid area. The left base, posteriorly, was dull, due to compression atelectasis of the lung. The abdomen was distended with fluid. The liver was palpable for a distance of one hand's breadth below the costal margin; it pulsated and a systolic bruit was heard over it; its edge was smooth. The spleen was palpable. Tests of the urine and blood gave negative results. The Wassermann reaction was negative.

This case is presented because it shows the classic features of a mitral and tricuspid rheumatic endocarditis with decompensation.

SELF-SELECTION OF DIET BY NEWLY WEANED INFANTS: AN EXPERIMENTAL STUDY (WITH LANTERN SLIDES). DR. CLARA M. DAVIS.

This article appeared in full in the October issue.

Annual Meeting, May 15, 1928

MAURICE BLATT, M.D., *President, Presiding*

ACUTE STREPTOCOCCIC SEPTICEMIA. DR. L. W. SAUER.

Richard K., aged 3½ years, was seen on April 24, at 1 p. m., because he had vomited several times in the latter part of the morning and had had two liquid evacuations. He had been indoors for several days with a slight cold in the head,

but in spite of the cold weather was taken on a long auto ride on April 23. He ate a hearty supper, was playful, slept most of the night and ate his breakfast as usual. About 10 a. m., he vomited for the first time, and after that was unable to retain anything. The temperature was 101 F.; respirations, 28; pulse rate, 92. He was not in pain and could sit, stand and lie down. Examination did not show any abnormal condition except a slight laryngitis and a distinct pharyngitis. The lungs, heart, abdomen, eyes, nose, ears and reflexes were found to be normal. The child appeared to have an infection of the upper respiratory tract involving mainly the larynx and pharynx.

At 8 p. m., he suddenly became much worse. He was unconscious; his color was extremely pale; respirations were 50, and very labored; the pulse was regular but slow and weak; the temperature was 103 F. Examination revealed numerous coarse and fine râles over the entire chest; reflexes were reduced; the pupils were large and responded slowly to light. The child vomited all evening, and the last vomitus contained brown blood. He was given 10 minims (0.62 cc.) of epinephrine chloride intramuscularly, and was taken to the hospital; on arrival, 150 cc. of dextrose Ringer's solution were given by hypodermoclysis. Within a few minutes, he had a series of short convulsions; at 10 p. m., about twelve hours after the onset of symptoms, he ceased breathing.

As the child was exceptionally well developed, overweight, and had had chronic eczema during the first two years of life, a tentative diagnosis of status thymico-lymphaticus with beginning capillary bronchitis was made.

Postmortem examination, performed by Dr. J. Leslie Williams, a few hours after death, revealed: acute pharyngitis, laryngitis, tracheitis and hypostatic congestion of both lungs; the thymus weighed 23 Gm.; the lymph glands of the mesentery, Peyers' patches and solitary follicles were rather large. There was no sign of pressure of the thymus on the trachea; nor was there any local edema. On the following day the throat culture and the plates of the heart blood showed innumerable streptococci of the hemolytic type. This discovery led to the diagnosis of acute streptococcic septicemia, following a streptococcic pharyngitis in a young child with lymphoid hyperplasia.

It is suggested that blood cultures be taken immediately after death in all cases of young children who die suddenly.

DISCUSSION

DR. SAUER: Examination of the spinal fluid was not made. The tonsils were rather large, cryptic and very red.

EARLY INTUSSUSCEPTION. DR. STANLEY GIBSON.

I have had the opportunity in the last two years to observe two cases of intussusception practically from the beginning; they came to operation earlier than usual.

CASE 1.—A girl, aged 5 months, was seen on Jan. 22, 1926. The mother telephoned me, at 10 p. m., that the child had developed suddenly a bluish pallor, cried and seemed to be in great pain. She said that she had just given the baby an enema and noticed some flakes of blood in the return.

When I arrived, the child's expression was pathognomonic of intussusception. I made a rectal examination and no blood returned. At intervals of from ten to fifteen minutes, attacks of severe colic recurred; during the intervals the child slept. In three quarters of an hour I gave another enema, and the return was mixed with blood and feces. At this time, I could palpate a tumor lying transversely in the midline. The first symptom had appeared at 10 p. m.; the child was taken to the hospital and operated on at 2 p. m., four hours after the first sign. A typical intussusception was found, which had progressed only about 2 inches (5 cm.).

CASE 2.—A healthy, breast-fed infant, aged 5 months, was seen in November, 1926. He suddenly gave a sharp cry, which was followed by evidence of severe shock. The first symptoms occurred at 9 p. m., and the infant was in a state of

profound shock when I arrived. Within an hour and a half after the first symptom, I gave an enema, and the return was mixed with blood. Within a short time, I was able to palpate the tumor. The child was taken to the hospital, and on arrival was asleep, passive and completely relaxed. A tumor could be felt and seen very plainly, extending across the abdomen. Operation was performed at 12:30 a. m., four hours after the first symptom, and the child made an uneventful recovery.

I think that the diagnosis can be made in the majority of these cases fifteen minutes after the onset, because of the characteristic appearance of the face and the acute colicky attacks. Within two hours after the onset, in this case, the entire symptomatology was present.

DISCUSSION

Dr. C. A. Aldrich: I saw a similar case about five years ago, and an unusual train of circumstances enabled me to see the patient immediately after the intussusception occurred. About 12:30 p. m., I made a routine call on an infant to observe its feeding. I left the house and made another call. When I returned to my office, there was a telephone call from the mother, who said that I had not been gone more than half an hour when the baby suddenly became violently ill. I returned, about an hour after my first call, and half an hour after the first symptom. The appearance of the infant was exactly as Dr. Gibson outlined. I made a rectal examination, and a typical red raspberry jam stool followed. Operation was performed three and a half hours after the onset of the symptoms, and an intussusception was found.

Dr. Joseph K. Calvin: I had a case of intussusception in which a barium sulphate enema was given before operation. Afterward, I saw two articles in the American Journal of Diseases of Children in which this procedure was used as a therapeutic as well as diagnostic measure. I think it is a good plan to give a barium enema under normal pressure. The intussusception in the case that I mentioned was reduced by this means during two attacks. Under ordinary pressure the intussusception slips back when the barium enters the colon. As this was a case with a history of three intussusceptions and two operations, with no further recurrence of the condition, it seems to me that it is a good plan to use the barium enema in the early stages of the condition. The progress of the enema can be followed under the fluoroscope, and if the intussusception is not reduced, very little time is lost.

Dr. George E. Baxter: I saw a case like this quite early, a few hours after the onset; I made the diagnosis of intussusception and started to the hospital with the child. On the way, the jarring of the automobile probably put the bowel back in place, because on arrival at the hospital, the physical conditions of the abdomen had changed, and the mass had entirely disappeared.

Dr. Julius H. Hess: During the past year and a half, I have seen three cases. One child had a large intestinal polyp which was discovered at the second operation. In another case, a similar condition was found at the third operation for intussusception. In every case of intussusception, search should be made for the underlying cause.

Dr. Gibson: Dr. Aldrich spoke of getting the raspberry jam stool on rectal examination in his case. I did not obtain that type of stool in either of the cases that I reported, but on giving an enema, I secured small amounts of blood mingled with the feces. That classic symptom, I think, comes later. In Dr. Aldrich's case, I believe that it was unusual to get so large an amount of blood so early.

Meckel's Diverticulum with Hemorrhage. Dr. M. G. Peterman.

This article was published in full in the September issue of the American Journal of Diseases of Children, p. 515.

DISCUSSION

DR. PETERMAN: In the cases in which the gastric mucosa has been found in the diverticulum, it has been considered the source of the bleeding. In the case which I reported, the gastric mucosa was intact; all the ulcers were in the Meckel's diverticulum, and at the base.

SEPTIC ENDOCARDITIS IN THE SECOND MONTH. MURAL ENDOCARDITIS. DR. GERARD N. KROST.

J. D., born on March 5, 1926, weighed at birth 9 pounds and 2 ounces (4,139 Gm.); on the ninth day, he weighed 8 pounds and 12 ounces (3,969 Gm.), and seemed in good condition. On the twelfth day of life, the infant developed a bullous impetigo, undoubtedly contracted at the hospital, as an infantile impetigo was prevalent in the nursery at the time of his birth. Abnormal symptoms did not develop until the afternoon of the eighteenth day, when the infant became listless and had a fever of septic type but of slight degree, from 102 to 103 F. Numerous petechial hemorrhages occurred over practically the whole body, particularly over the chest and abdomen. On the twentieth day of life, the baby was admitted to the hospital. He was covered with many small pustules, a fair number of bullae and innumerable closely grouped petechial hemorrhages on the chest and abdomen. He was apathetic and could not be aroused. He was feeble, and attempts to nurse him at the breast and to feed him from the bottle were unsuccessful. The throat and ears were normal. The heart and lungs also were normal. The abdomen showed a moderate distention. Laboratory examinations and blood culture were not made.

The infant was fed by gavage, every three hours, with a protein milk formula. An intraperitoneal transfusion of 40 cc. of the mother's blood was given. Feedings had to be given by gavage for the first four days in the hospital. The skin petechiae began to fade; the baby appeared stronger, and cried vigorously; his temperature became normal on the fifth day in the hospital. He was discharged on the twelfth day, apparently in good condition and with a normal temperature; he was able to take the feedings well. The temperature remained normal, and the baby gained in weight; the stools were good; apparently entire recovery from sepsis had been made.

On the forty-third day of life, the infant suddenly became prostrated; he was very pale, and his respirations were shallow, almost ceasing. By the time the physician reached the house and examined him, the child looked and acted almost normal, except for severe distention of the abdomen. He was readmitted to the hospital at 1:50 p. m. Other than an appreciable abdominal distention, which did not respond to enemas, symptoms were not noted. The heart and lungs were examined, but disclosed nothing abnormal. Evidence of petechial hemorrhages of the skin were still present. The baby acted vigorous and took his feedings of 3 ounces (89 cc.) very well. At 11:20 p. m., the infant suddenly turned very pale, first became dyspneic, then had shallow respirations and suddenly died.

Postmortem examination on April 28, 1926, revealed the pleural cavities free from fluid and adhesions. Both lungs had smooth pleural surfaces on which an occasional petechial hemorrhage was visible. The lungs were normal. The thymus was normal. The heart was somewhat larger than usual for the size of the infant. The pericardial sac contained a few cubic centimeters of clear, straw-colored fluid, and its lining was smooth. Numerous petechial subendocardial hemorrhages were present, especially about the upper anterior area of both ventricles. On section, the myocardium was light pinkish red, with occasional light yellow mottlings. The endocardium of the right chamber and valve was free from change. The mitral valves showed numerous small glistening red nodular masses, 1 mm. in diameter, located in a row near the free margin of the cusps; they were rounded and firm and did not brush off easily. A few masses similar in appearance but somewhat larger were found on the lateral surface of the left auricle. The aortic valves were free from change. The coronary orifice appeared patent. The foramen ovale was rather freely patent, although the membrane was sufficient for closure.

The liver was of ordinary size. There were multiple hemorrhages into the parenchyma of the liver. The spleen was moderately enlarged. There was acute hyperplasia. The kidneys, bladder, gastro-intestinal tract and pancreas were normal.

The anatomic diagnosis revealed: (1) subacute verrucous endocarditis of the mitral valve and left auricle; (2) acute parenchymatous degeneration of the myocardium; (3) petechial hemorrhage into the parenchyma of the liver; (4) petechial hemorrhages into the skin; (5) acute hyperplasia of the spleen; (6) patent foramen ovale.

The immediate cause of death was not revealed by the autopsy. It was suggested that an embolus may have lodged in the brain (not examined), or in a coronary artery, where it was not found. The infant apparently had made a complete recovery from its sepsis and died from the result of the damage done to the heart or from an embolus formed from the septic bodies on the endocardium.

CASE 2.—L. B., a girl, aged about 8 years, white, entered the hospital on Aug. 10, 1926, with a history of weakness and shortness of breath on exertion for two months and swelling of the feet and the face for three weeks. The symptoms developed gradually in severity, until the patient recently had been forced to remain in bed. One year before admission to the hospital, she had had rheumatism and heart trouble and had remained in bed for three months. She had had whooping cough at 5 months of age and measles at 3 years. There was no history of rheumatism in any other members of the family. Physical examination revealed a dyspneic and cyanotic child, with cyanotic lips; her teeth were in good condition; her tonsils were small but cryptic without evidence of acute inflammation. There was a heaving apex beat, visible in the axilla, in the fifth interspace. The skin of the chest and abdomen had a glossy appearance, due to edema. There was no retraction or Broadbent's sign to be seen. Posteriorly over the right side, and extending to the angle of the scapula, there was dulness of the lungs and there were suppressed breath sounds. On the left side, just at the angle of the scapula, the breath sounds increased.

On the left border of the heart, the apex beat was in the anterior axillary line. On the right border over the base, there was an increased area of dulness which extended toward the right, in the third interspace, 5 cm. from the sternum. A systolic thrill could be palpated over the entire precordium. A marked systolic and a presystolic murmur was heard at the apex. The heart tones at the base were so muffled that it was almost impossible to hear them.

Ascites was present. The liver was enlarged to a hand's breadth below the costal margin and was tender. There was edema of the extremities and of the sacral region.

The diagnosis was mitral regurgitation and mitral stenosis, with active adhesive pericarditis and right hydrothorax ascites.

Laboratory tests, made on the patient's entrance to the hospital, showed a trace of albumin in the urine but no casts, only a few red cells and 100 white cells per one-eighth power field. The blood count showed 5,240,000 red cells, 24,600 white cells and 85 per cent hemoglobin. A roentgenogram of the heart revealed the heart shadow as the triangular, appreciably enlarged area characteristic of the pericarditis with effusion. Both of the cardiophrenic angles were obliterated. The pulmonary fields were clear. Roentgen-ray examination showed the nasal sinuses to be normal.

The patient remained in the hospital until death occurred, on the one hundred and twenty-second day after entrance. The highest temperature recorded was 102.2 F., rectal. The rectal temperature rose above 99.6 F. during only fourteen of the 122 days in the hospital; during the first eighteen days the temperature did not rise above 99.6 F. This comment is made in the light of some criteria recently given us for judging the importance of temperature in the determination of quiescence of infection. The pulse rate varied from 100 to 120. The urine was normal, except for a trace of albumin. There was no essential change in the cardiac observations at any time. The patient appeared moderately well from

about the tenth to the seventy-fifth day. About the seventy-fifth day, edema began to recur and the patient looked worse, slept a great deal and had frequent attacks of precardial pain. At this time, her pulse became irregular and it was thought that auricular fibrillation was occurring. An electrocardiogram tracing showed a normal mechanism interrupted by many premature ventricular contractions. The voltage was very low in the first lead. Death occurred from a gradual heart failure on the one hundred and twenty-second day in the hospital.

Postmortem examination showed a girl, aged about 8 years, white and fairly well nourished. The skin had no petechiae. The abdominal cavity contained 350 cc. of a straw colored fluid. The left pleural cavity contained 30 cc., and the right pleural cavity 15 cc. of a straw colored fluid. The pericardium was thickened, grayish and contained 25 cc. of straw colored fluid. Pericardial adhesions were not present. The heart was larger than that of an adult male fist and compressed the right lung. The visceral pericardium showed many petechiae, especially near the auriculoventricular junction. The right ventricle was about 3 mm. thick, and the walls were smooth. The pulmonary orifices and the valves were normal. The right auricle was of normal thickness, and the lining was smooth. The left ventricular wall was about from 10 to 12 mm. thick, and the lining was smooth except for fibrous patches near the base of the aorta. The aortic cusps and orifice were normal. The left auricle wall was thickened. The endocardium was rough, grayish and opaque, studied with innumerable vegetations. The mitral valve was 7 cm. in circumference and was thickened; there were few vegetations. The myocardium showed occasional fibrous patches throughout. All the lobes of the lung were crepitant except the lower lobe of the left lung which showed a dark area, which had the appearance of hemorrhage. In the abdomen, the parietal and visceral peritoneum were smooth and glistening; the liver was markedly enlarged, and five finger breadths below the costal margin. The kidneys were normal. The ureters, bladder and gastro-intestinal tract were all normal. The mesentery showed numerous small petechial hemorrhages throughout.

The anatomic diagnosis was: (1) hypertrophy and dilatation of the heart, (2) chronic fibrous endocarditis of the mitral valve (moderate) and of the lining of the left auricle (extreme), (3) chronic fibrous myocarditis, (4) considerable passive hyperemia of the lungs, liver and spleen, (5) anasarca and (6) petechial hemorrhages into the pericardium.

The unusual features of this case are the marked involvement of the endocardium of the left muscle and the slight involvement of the valvular endocardium. The error made in this diagnosis is of clinical interest. It reverses the frequent error of diagnosing an adhesive pericarditis as a valvular endocarditis because of the murmurs present in an adhesive pericarditis from the weakened myocardium, allowing regurgitation through a stretched valve.

Diphtheria Three Weeks After Injection of Toxin Antitoxin in Child Sensitive to Serum. Dr. Iris Chamberlain.

Margaret K., aged 5½ years, did not give a history of eczema or asthma, and had never received serum previously. The Schick test gave positive results, and she was given three injections of toxin antitoxin (containing horse serum) at seven day intervals. Neither a local nor a general reaction occurred.

Twenty-four days after the last injection, I was called to see the child; she had vomited and had not felt well, but was not thought to be particularly ill. Her temperature was 100.5 F., and her throat was slightly infected, with a patch of grayish exudate covering one third of the left tonsil. A culture, read twelve hours later, showed *B. diphtheriae* in practically pure cultures. With the commercial package syringe, a little antitoxin was injected just under the skin of the left thigh. An immediate reaction followed; in five minutes, the wheal was 6 inches (15.24 cm.) in diameter. The child became restless, complained of being uncomfortable, the respirations were increased, but there was no cyanosis. She received 5 minims (0.31 cc.) of 1:1,000 epinephrine at once. Three doses of antitoxin, given with a graduated hypodermic syringe, were administered at thirty

minute intervals: the first, 1 minim (0.062 cc.); the second, 4 minims (0.25 cc.); the third, 1 cc. Following each of these injections, 5 minims (0.31 cc.) of 1:1,000 epinephrine was given, as there was a prompt and rather appreciable local reaction. The wheals were practically identical in intensity, and varied from 6 to 8 inches (15.24 to 20.32 cm.) in diameter. Thirty minutes after the last subcutaneous injection, a fourth injection of 1 cc. of antitoxin was given intramuscularly. The child continued to be restless, with the respirations only moderately increased, and the pulse regular and full. Thirty minutes later, the remainder of the dose, 20,000 units, was given intramuscularly. The following morning the temperature was 104 F. The child complained bitterly of the pressure from the bed clothes, or of a change in position. There was a generalized urticarial eruption; the hands and feet were edematous; the right knee was swollen, and movement of the left hip joint was painful; there were painful indurations about each point where the antitoxin had been injected. The face was puffy. Itching was severe. Ten minims (0.62 cc.) of epinephrine produced little, if any, relief. Ephedrine sulphate, in doses of 5 minims (0.31 cc.), was administered every four hours, with 15 grains (0.98 Gm.) of calcium chloride. Calamine lotion, containing 1 per cent phenol, was used locally; the right knee was kept in a wet dressing of magnesium sulphate. The urine was heavily loaded with albumin.

Forty-eight hours after the antitoxin was given, the membrane had practically disappeared. The temperature fell slowly, reaching normal on the third day. The swelling of the face, hands and feet persisted for twenty-four hours, that of the knees for seventy-two hours, and movement was uncomfortable for seventy-two hours. The urticaria disappeared gradually in the course of five days. The ephedrine was discontinued after forty-eight hours, but the calcium continued to be used three times daily for ten days. The cultures remained positive for nine days, but there were no further reactions. Sixty days later, the Schick test gave negative results; the cutaneous reaction to horse serum was positive.

DISCUSSION

DR. GERARD N. KROST: I wish to know why this child was tested for sensitivity.

I would also like to know if any one here has had any clinical experience with the use of atropine to prevent the anaphylactic reaction. In experiments on animals, the administration of a large dose of atropine as a prophylactic measure will prevent death. In a recent case of a child who had received injections of antitoxin, and who had a history of being asthmatic, I gave a dose of one-hundredth grain (0.65 mg.) of atropine, having in mind the experimental work in anaphylactic animals.

DR. JOSEPH K. CALVIN: I think we will not have this sort of difficulty in the future, as a very reliable sheep serum is now prepared for use. I think everyone has had the experience of having diphtheria developed before the immunity was entirely established, but with the use of the sheep serum that is being put out now commercially, the danger of anaphylaxis by subsequent injections of horse serum is practically obviated.

DR. JULIUS HESS: I should like to know whether any one has had any experience with the use of toxoid.

DR. PHILIP ROSENBLUM: I gave toxoid to the children of a physician because he wanted to avoid the reaction. I tested them a month afterward and found that a Schick test produced negative results; whereas the reaction before had been positive. I think that in Canada they use the toxoid almost exclusively; the only experience I have had, however, was in these two cases, and it seemed to work well.

DR. CHAMBERLAIN: It was our rule that each injection of the antitoxin should be preceded by a subcutaneous injection.

The reason atropine was not given was because the child had a distinct physiologic reaction after receiving a dose of belladonna during an infection of the upper respiratory tract.

CONGENITAL ESOPHAGEAL STENOSIS. DR. JOHANNA HEUMANN.

For a girl, aged 3, a diagnosis of congenital stenosis of the esophagus was made, based on a history of regurgitation of all solid food, with retention of liquids, and on the disclosure of a marked narrowing of the lower portion of the esophagus with dilatation above by the x-rays. The narrowing was less than 15 mm. in circumference. After twenty-five dilatations, the esophagus had the normal circumference of 26 mm. The child made a complete clinical recovery; during the subsequent two years, the stenosis did not show any tendency to recur.

The eighty cases found in the literature were reviewed; fifteen could not be verified. Of the ten museum specimens reported on by Keith, one was identical with the case reported by Home. In the sixty-two cases which were studied, twenty-one of the patients were American, twelve English, ten French, six German, six Scotch, two Swiss, two Swedish, one each Danish, Irish and Dutch. Thirty-one were male, twenty-five female and in six cases the sex was unknown. The youngest patient was 5 days old; the oldest, 84 years. Sixty-one were white, and one was colored. Most of the stenoses were located at the lower portion of the esophagus. There was a record of postmortem examination in twenty cases. Fifty-five patients had shown typical symptoms since birth or since weaning. In four the symptoms were mild; in two they were of comparatively short duration. Twenty had severe attacks in which they vomited even liquids. In twenty-three, diagnostic bougies were passed; in six, sounds, and in 1, a catheter. In twenty-seven, the diagnosis was confirmed by x-ray examination and in fifteen by esophagoscopy. Twenty-six patients died; in fourteen of the cases the cause of death was starvation, directly attributable to the stenosis; in seven, death was caused by pneumonia; in five, it was attributable to gastrostomy and in one to perforation.

Thirty-one of the sixty-two patients were cured and dilatation was performed successfully on twenty-four. Bougies without a guilding string were used in most cases. In three, bougies were used with temporary relief; in two, bougies were passed unsuccessfully; in one, a bougie was passed, causing perforation and death. In five children, gastrostomy was performed successfully, and in five it resulted in death. In twenty, treatment was not attempted, although in thirteen of these the stenosis had been diagnosed.

FRAGILITY AND DEFICIENCY HEALING OF BONE IN POLIOMYELITIS. DR. I. HARRISON TUMPEER.

Poliomyelitis signifies an acute illness in a child who develops weakened extremities, then paralyses, absent reflexes, and the reaction of degeneration and shriveling. The shriveling involves all of the tissue, including the bone, a fact which is frequently overlooked.

The neurologist explains that the atrophy of the bone is due to the absence of the trophic influence of the anterior horn cells which are destroyed in this disease. The surgeon, on the other hand, explains the changes of the bone on the basis of nonuse. To throw some light on this question, the following more formal data are submitted.

The bone changes in an extremity paralyzed by poliomyelitis were accurately described even before the x-ray came into use, and have been abundantly confirmed since that time. They consist, for the most part, in generalized atrophy, smoothness of surface, uniformity of thickness and relative transparency, due to the fact that the muscular actions which cause the tendon grooves and irregularities at the points of insertion are no longer active. Shortening is less marked than the other changes and points to the fact that the process is a diaphyseal rather than an epiphyseal process.

The point of view of the neurologist has been attacked through experimental work in animals and clinical observations in man. In experiments, atrophy occurred whether nonuse was produced by paralysis of the nerve, injuries to the joint or simple fixation in a plaster cast. The atrophy was directly proportional to a degree of nonuse. Of particular interest was the fact that regeneration was not

affected, so that a section removed from a paralyzed member was liable when transplanted to the normal extremity.

Two cases are reported. The first was that of a girl, aged 5 years, with complete paralysis of one arm from poliomyelitis which had occurred four years before. Following a relatively slight trauma, there was a complete fracture of the humerus in the affected arm. The x-ray picture also showed the usual atrophic changes. Healing occurred; two months later, there was another fracture at the same site, following a minor trauma. Fractures occurred repeatedly thereafter, but healing and callus formation always followed. The course of this condition was confirmed by x-ray observations.

The second case was that of a boy, aged 10, who suffered a fracture of the femur following an automobile accident. Poliomyelitis had affected this extremity six years before. There was marked overriding of the fragments, and repeated efforts at closed reduction were unsuccessful. An open reduction was performed, and a graft from the normal tibia was used as an inlay to bring the fragments in line. There were the usual atrophic changes in the affected femur and a relatively weak callus before an open operation was performed. Subsequent x-ray observations showed that callus formation took place, and the bone graft appeared to be attached by the callus.

These cases indicate that callus formation and regeneration does occur, despite the nerve lesion, and confirms the deductions from experimental observations in animals on these points. The explanation of the fragility of the bone, even after healing, lies in the fact that functional stimulus was lacking because of the paralysis. The functional stimulus is important in maintaining the normal condition of bone, and is particularly important in the healing of fractures as the callus produced is not merely a cement, but in shape and structure is adapted to the use for which it is intended.

The result of these observations indicates that atrophy of the bone in poliomyelitis is indistinguishable from atrophy of the bone resulting from nonuse. The atrophic bones are usually fractured. Callus formation occurs, but produces a weak union because the functional stimulus is absent.

NORTHWESTERN PEDIATRIC SOCIETY

July 14, 1928

W. R. SHANNON, M.D., *President, in the Chair*

THE FILTRATION OF COLON BACILLI THROUGH LIVING MEMBRANE. Presented by DR. S. AMBERG.

These experiments were done on rabbits in a state of narcosis produced by urethrane. Sodium chloride solution was used as filtering fluid. While the experiment was in progress, one could see pulsation in some vessels and had other evidence that the membrane was living while filtration went on. The filtrate could usually be obtained free from blood so that it did not give a test with guaiac or benzidine. When a suspension of colon bacilli in solution was placed on the membrane, the colon bacilli passed through readily and could be found in the filtrate.

Suspensions of colon bacilli were injected intravenously and saline placed on the membrane to see whether bacteria came through; none of them did in from twenty to thirty minutes, whereas a culture taken from the blood showed large numbers of colon bacilli. Blood obtained by nicking the vessels in the membrane contained colon bacilli. Then the experiment was varied, and under aseptic precautions the abdomen was opened and kaolin applied to the mesentery, setting up an inflammation. The next day this mesentery was used as a filter. It was more difficult to get blood-free filtrate because the vessels were rather easily injured, but even if there was blood in the filtrate at first, it disappeared later. When

colon bacilli were injected intravenously, they did not appear in the filtrate within the time limits except when there was definite injury to the blood vessels.

Under the conditions of the experiment, it seems that bacteria injected into the circulation in large numbers do not penetrate into the tissue in such a manner as to become readily demonstrable. The observations are in accord with results obtained by Dr. Herrmann who injected bacterial suspensions intraperitoneally and was unable to show their presence in the blood stream. The experiments have thus far been qualitative. In order to know more about their significance, quantitative experiments have to be done. If one accepts the theory of the discharge of bacteria from foci into the blood stream and from there into the tissues, these experimental results suggest that, at times at least, an additional factor such as injury to the capillaries must be taken into consideration.

DISCUSSION

DR. SWANSON (Minneapolis): I should like to ask if stretching of the membrane makes any difference in the rate of filtration.

DR. AMBERG: We have not done experiments on this. The pressure is really very low.

REPORT OF TWO CASES OF APLASTIC ANEMIA. Presented by DR. A. S. HURT, JR.

A boy, aged 13, was admitted to the clinic on March 22, 1928, because of anemia. In May, 1927, it had been noted that he became breathless while at play and that he was pale. He had also had slight bleeding from hemorrhoids. Blood had been found in the stools on three occasions in January, 1928. He had been given mild mercurous chloride for colitis for about six weeks. He had been bruising easily and was admitted with a diagnosis of purpura hemorrhagica.

He was well developed and well nourished with marked pallor of the skin and mucous membranes. There was some edema. Stomatitis was present. The spleen was not palpable. Examination of the blood showed: 17 per cent hemoglobin, 820,000 erythrocytes and 1,800 leukocytes with 65 per cent lymphocytes, 0.5 per cent large mononuclears, 4.5 per cent transitionals, 29 per cent neutrophils and 1 per cent eosinophils. There was moderate anisocytosis and slight poikilocytosis. The platelets numbered 136,000. The coagulation time ranged from six and a half to ten minutes. The bleeding time was sixty minutes. There was a marked increase in the fragility of the red blood cells. There was no clot retraction in sixteen hours. The prothrombin time was normal.

During seventy-two days in the hospital he was given fourteen transfusions and a diet high in vitamin and iron, and he received liver extract. The hemoglobin rose to 54 per cent, erythrocytes to 2,500,000 and leukocytes to 3,000. The highest reticulated cell count was 1.2 per cent and the lowest 0.1 per cent. A week before death, which occurred on June 2, he had severe nasal hemorrhage, and the hemoglobin dropped to 20 per cent. Further transfusions were ineffectual. Rectal oozing of blood appeared.

At necropsy a diagnosis of aplastic anemia and cerebral hemorrhage was made. Petechial hemorrhages into the skin, brain, lungs, heart, kidneys and mucous and serous surfaces throughout the body were found. The bone marrow of the right femur had the appearance of fat tissue without brick-red areas. The spleen weighed 182 Gm. Microscopic examination of bone marrow showed little hematopoietic tissue. A few inactive islands of leukoblastic tissue, but no erythroblastic tissue, were demonstrated.

A girl, aged 4, came to the clinic in May, 1925. One week before the mother had noted large, subcutaneous blue spots over the surface of the body. Small red petechiae had appeared on the legs and neck. There was no history of injury or bleeding.

Gingivitis and dental caries were present. Subcutaneous hemorrhages were found on the arms, legs, back and abdomen. Numerous petechiae were present on the soft and hard palate. The spleen was not palpable. The urine showed

red blood cells, graded 5 on the basis of 1 to 5. The hemoglobin content was 43 per cent; the erythrocytes numbered 2,920,000 and the leukocytes 2,900; there was a normal differential count. The coagulation time was three minutes. The bleeding time was seven minutes. The prothrombin time was prolonged. Platelets numbered 98,000.

Four transfusions were given, and the spleen was removed on June 25, 1925. Petechiae and purpuric spots continued to appear, and there was some abdominal pain. Four more transfusions were given. Tonsillectomy was performed. It was almost impossible to stop hemorrhage by ligatures. Two days later transfusion was necessary. Death occurred in three days. The clinical diagnosis was purpura hemorrhagica.

At the time the pathologists concurred in the diagnosis, but since the bone marrow of the femur was aplastic and pale yellow. In retrospect, the diagnosis in this second case may be questioned. Aplastic anemia certainly should have been considered from the data at hand and the clinical course. In purpura one would expect to find a high white blood cell count, whereas in aplastic anemia it is usually extremely low. The platelets are low in both conditions, but lower in hemorrhagic purpura than in aplastic anemia. In aplastic anemia reticulated cells are rather low in number, and in hemorrhagic purpura they are usually rather high.

DISCUSSION

DR. SCHLUTZ: I should like to ask Dr. Hurt whether there was any noticeable difference in the improvement when the liver extract was added. We had one case at the University Hospital, five or ten months ago, in which there was a clear picture of aplastic anemia. Transfusions were resorted to, and the child was showing fairly steady improvement but seemed to do definitely better when fetal liver extract was added. There was always a doubt in my mind as to whether the improvement came about without it, but it surely was striking. I do not know to what extent fetal liver extract is used here, but I should like to know whether there was any difference in the blood picture after the administration of the liver extract.

DR. HELMHOLZ: It is of further interest that in the second case, in which the diagnosis was hemorrhagic purpura, the patient was sent to the hospital for splenectomy. Although the two conditions seem different, they may resemble each other closely. Only recently Conner reported cases of this type in which the differential diagnosis was extremely difficult and emphasized the point that Dr. Hurt has brought out in differentiating the two conditions.

DR. AMBERG: With regard to the question of Dr. Schlutz, we used common liver preparation without results. We tried to obtain fetal liver extract but could not do so.

THE PATHOLOGY OF PYELITIS: EXPERIMENTAL AND IN MAN. Presented by DR. R. L. J. KENNEDY.

This paper will be published in full later.

DISCUSSION

DR. HELMHOLZ: I should like to call your attention to the point Dr. Kennedy made with regard to obtaining material from a human being in order to get an idea of how this process starts. I doubt whether we shall be able to get pathologic material from pyelitis of less than ten or fifteen days' duration. The first slide showed extensive involvement of the kidney. If this is the earliest picture of pyelitis that we can expect to see in kidneys of human beings, we must depend on our experimental material to determine what the earliest changes are. It is impossible to say whether that lesion began in the cortex and spread down or up. In the experimental material the tendency of the infection is to start at the cortex and work down. The circulation of the kidney is such that healing in the renal

parenchyma is much faster than in the peripelvic tissue. The tendency is for the lesion in the parenchyma to heal rapidly and for the lesion in the peripelvic tissue to resolve slowly. These studies should give a fairly good idea of the development of these processes and by following through this rabbit material I think we shall be able to interpret the human material as it fits into the series with rabbits.

DR. SWANSON: Were there any lesions in the other internal organs, as well as in the kidneys, after the injection of colon bacilli.

DR. KENNEDY: In one or two instances there was a suppurative process. I think there was one around the appendix and one in which the process developed in the lung, but there was exceptional freedom from lesions in the organs from this particular strain of the organism. I may say that Dr. Helmholz has reported this. It has been difficult to find organisms which will perform satisfactorily.

DR. SHANNON: I think we might be able to help Dr. Helmholz and Dr. Kennedy if we kept it in mind. Somebody is going to see a case that will be of value in this instance.

PELLAGRA IN A PATIENT ON THE KETOGENIC DIET. Presented by DR. R. B. STEVENS.

A girl, aged 5, came to the clinic in October, 1925, for treatment of epileptic seizures. Physical and laboratory examinations showed nothing unusual. The child was put on the ketogenic diet and returned home where she was unmanageable and at times broke the dietary restrictions. While ketosis was present, she was fairly free from attacks. In June, 1927, a rash appeared on her arms, hands and feet. This lasted for a month and then disappeared completely. She remained on the ketogenic diet with occasional lapses. In June, 1928, she again came to the clinic with a rash on the hands and neck which had appeared about nine weeks before between the forefinger and thumb on each hand, and then had extended over the palm and back of the hand and to the neck.

Physical examination showed a dry, harsh appearance of the skin of the face. There was thick desquamation of the skin of the neck. The back of the hands had a glovelike thickening which extended over the wrists. The tongue was desquamating with large, red follicles. The teeth were in excellent condition. The blood calcium was 11 mg. A ketogenic diet, high in vitamin content, was arranged. The pellagra improved noticeably while she was here.

DISCUSSION

DR. ANDERSON: I have recently placed a 10 months old patient on the ketogenic diet. The child had been having attacks of petit mal type since 2 or 3 months of age. Phenobarbital and bromides had not controlled the attacks. The ketogenic diet, which was established in this case without difficulty, has given a good result. For the past three months the child has not had convulsions.

DR. SCHLUTZ: Was there much loss in weight?

DR. STEVENS: No.

DR. CRICHFIELD: Was the pigmentation marked?

DR. HELMHOLZ: It was difficult to tell how much was pigmentation and how much was actually due to treatment, for a number of ointments had been used.

THREE CASES OF GHON TUBERCLE. Presented by DR. V. B. DOWLER.

The first case was in a girl, aged 10, who was adopted so that detailed account of her early history was not available. She was known to have had chickenpox, measles and mumps. She was brought to the clinic for general examination. She was somewhat underweight. Her tonsils were infected, and she had flat feet, graded 3 on the basis of 4. The blood count was normal. Pirquet and Mantoux reactions were positive. A roentgenogram of the chest showed Ghon tubercle.

The second case was in a girl, aged 8. There was nothing unusual in the family history. She had had measles at 2 years of age. She had had tonsillitis. At 4 years of age she had fallen and fractured a rib. Swelling had occurred at

the site of fracture and operation was advised. Following the operation there had been drainage for several weeks, but after a second incision there had been complete recovery. The weight and height of the patient were normal. The tonsils and adenoids were infected. The blood and urine were normal. Pirquet and Mantoux reactions were positive. A roentgenogram of the chest revealed healed tuberculosis of the right upper apex and Ghon tubercle. The third case was that of a girl, aged 11, whose father had died of tuberculosis when she was 6 years of age. Her past history was negative. She was presented with a complaint of headaches, pain and slight stiffness of various joints. The height and weight were normal. The Pirquet and Mantoux reactions were positive. A diagnosis of Ghon tubercle with calcification in the right apex was confirmed by roentgen-ray examination.

It is interesting to note that the patients in all three cases were girls and that the diagnosis was confirmed by the roentgen ray. The condition is comparatively rare. Two of these cases, the first seen here in seven years, were presented in one week.

DISCUSSION

DR. HELMHOLZ: I should like to ask how frequently other members see these Ghon tubercles.

DR. SCHLUTZ: I think such cases are rare. I recall only one considered to be such on x-ray observations. The child had a positive tuberculin test. In this case the patient recovered.

DR. SHANNON: I think I have seen one case in a small child. The x-ray diagnosis was that at least. The child recovered.

THE PRESENCE OF STREPTOCOCCUS HEMOLYTICUS IN NASOPHARYNGEAL CULTURES IN HEALTHY CHILDREN AND IN THOSE SUFFERING FROM INFECTIONS OF THE UPPER RESPIRATORY TRACT. Presented by DR. H. F. HELMHOLZ.

This paper will be published in full later.

DISCUSSION

DR. SCHLUTZ: It is an extremely interesting study. I agree with Dr. Helmholz that as yet little can be said of the significance of the observations. It is strange that an organism at times so dangerous is most of the time apparently harmless. I do not know whether there is any way in which the effect of the environment on the person and the organism could be studied. There may also be a question of the person's immunity at the time. It is surprising to find that the organism is so prevalent at all times of the year apparently without harmful effect.

DR. SHANNON: It seems to me that this is an extremely interesting study. I am sure that I thought the organism was even more common during epidemics such as occurred during the past year. I thought that this epidemic was due to a hemolytic streptococcus, and I imagined that the percentage of positive cultures taken throughout the city of St. Paul would have been extremely large. In fact, I am surprised that the percentage is so small. It illustrates how little an idea one has about the spread of infection.

RECURRING FUNCTIONAL MURMUR IN A CASE OF ANEMIA. Presented by DR. S. W. ADLER.

A boy, aged 6 years, was brought to the clinic because of progressive weakness and slowly developing anemia. He weighed 45 pounds (20.4 Kg.) and was definitely undernourished. Marked pallor of the skin and mucous membranes was present. The spleen was palpable three fingerbreadths below the costal margin. The hemoglobin was 23 per cent and the leukocytes 15,000. The platelets numbered 40,000. Other noteworthy variations in the blood picture were not found. At the first examination several observers noted a "to and fro" murmur to the left of the

sternum in the second and third interspaces. The characteristic soft blowing murmur followed immediately on the second sound. Because of the suggested endocarditis with aortic involvement, the patient was hospitalized.

On the fourth day of observation he received a transfusion of 500 cc. of citrated blood. Two days later the murmur at the base was not audible; the systolic murmur alone persisted. The hemoglobin rose to 40 per cent, and the leukocyte count was within normal limits. A second transfusion was given two weeks later, though the hemoglobin was only 34 per cent. After removal of several infected deciduous teeth he returned home, since he was improving generally.

Readmission was necessary three weeks later. Following transfusion the hemoglobin rose to 51 per cent. The diastolic basal murmur had not been heard since the first examination. It was felt to be undoubtedly functional. The loud rough systolic murmur with maximal intensity at the base persisted. It was less distinctly heard at the apex and not beyond. During the next three weeks the hemoglobin dropped to 31 per cent, and again the consultant distinctly heard the diastolic murmur in the same place. Transfusion of 500 cc. of blood was followed by disappearance of the diastolic murmur. Four days later one observer did not hear the murmur while one consultant heard it indistinctly but on the next day could not distinguish it. Soon after this, bronchitis, pneumonia, pleurisy with effusion, glossitis, stomatitis and pericarditis developed. Repeated transfusions together with application of radium to the spleen and lesions of the mouth did not check the unfavorable course. He died at home one month after leaving the hospital.

The case was observed nearly continuously for six months and was finally classified among the cases of so-called splenic anemia. The important feature is the relation of the basal diastolic murmur to the anemia on at least two occasions, with disappearance after the condition of the blood had been improved by transfusions.

Few references have been made to this particular functional murmur. Most standard textbooks on cardiology do not mention it. Cabot and Locke reviewed the literature and compiled a list of cases in which there were diastolic murmurs at the base suggesting aortic lesion but in which valve lesions were not found at necropsy. Of the various classes outlined by them one was associated with profound anemia. These reports were on adults, and do not speak of any transient character of the murmur. Cabot feels that functional diastolic murmurs are rare and are usually associated with profound anemia and hypertension. These observers, as well as Fisher, whose report is on children, call attention to another class of functional diastolic murmurs, basal in location, due to pericardial adhesions, and referred to as cardiorespiratory in type. They are described as rather infrequent, but no mention is made of intermittency.

Morse, in 1924, described this phenomenon and pointed out its "rather frequent association with severe anaemias of infancy and childhood." It accompanies cases both with and without cardiac hypertrophy. If the anemia is profound, the murmur may be associated with most of the signs usually ascribed to aortic regurgitation. There may be the pistol shot sound in the groin and the double murmur in the region known as Duroziez's sign. According to Morse, the changes are due to lowered vasomotor tone as a direct result of the anemia, together with relative insufficiency of the aorta from dilatation. He discredits the explanation based on a thinning of the blood. That it is not an organic change is proved by the fact that the murmur varies with the intensity of the anemia, disappearing when the anemia is relieved.

Additional evidence of postmortem absence of changes in the valves is desirable to assist in bearing out Morse's contention. In this case the physician at home made a postmortem examination and reported that the heart was about twice normal size with thickened, adherent pericardium. The valves did not appear to be involved.

DISCUSSION

DR. SHANNON: I should like to report a case of mine in which exactly this thing occurred. A boy, aged 9, first came to see me because he had vomited blood

on two or three occasions a week previously. At the time I saw him he had a profound anemia and a moderate fever. He had a widely dilated heart with numerous murmurs. There was a mitral presystolic murmur, a mitral systolic murmur and a systolic murmur over the base with considerable evidence of dilatation. I made a diagnosis of general pericarditis, and the boy was hospitalized on a cardiac regimen. He recovered without further bleeding in two or three months. The heart returned to normal, and I felt there were no signs of cardiac abnormality.

Two years later the father called me and said the boy had suddenly vomited blood again. I saw him after the first hemorrhage; the heart was normal with no signs of decompensation; so the hemorrhage had to be explained on another basis. The boy continued to vomit blood, and as the anemia became more severe one after another of the cardiac murmurs appeared; the systolic over the mitral, and a presystolic murmur in that region, and systolic and diastolic murmurs over the base accompanied by diastolic murmur and Duróziez's sign. The boy was given transfusions; following these the murmur disappeared and he was in fairly good shape for operation. My diagnosis was bleeding duodenal ulcer, although I did not know just what we were dealing with; however, we had some evidence of deformity as shown by the x-ray picture.

At operation he was found to have cirrhosis of the liver and bleeding from esophageal varicosity. The boy developed peritonitis and died. We were fortunate in securing a postmortem examination and found the heart perfectly normal except for lesions which accompany severe anemia.

DR. SCHLUTZ: How old was the boy when the first hemorrhage occurred?

DR. SHANNON: He was 9 years of age. He was 11 years old when he died. The spleen was much enlarged. I thought of the possibility of Banti's disease, but Dr. Bell did not think it was that. The spleen was not palpable before.

THE EFFECT OF OXYGEN ON HEALING IN CAPILLARY BRONCHITIS (ABSTRACT OF CASE TO BE REPORTED IN DETAIL LATER). Presented by DR. R. E. NUTTING.

A male infant, aged 11 months, with capillary bronchitis was treated for twenty-six days in the oxygen tent. Cyanosis and other signs of collapse appeared immediately on any attempt to remove him from the tent. There were no marked physical signs in the chest until ten days before death when dulness and tubular breathing were noted over the lower posterior portion.

At necropsy the lungs were firm in consistency especially in the lower and posterior portions. Firm discrete nodules could be palpated in the lung substance. Microscopic section showed the small bronchi and bronchioles to be filled with fibrous tissue to a point of occlusion in many places. Death was explained by this unusual organization process.

DISCUSSION

DR. CRICHFIELD: The picture is somewhat suggestive of syphilitic pneumonia with connective tissue increase. Was syphilis ruled out?

DR. NUTTING: So far as I know a Wassermann test was not done.

DR. ANDERSON: I wonder if capillary bronchitis is not much more common than the old textbooks would lead us to believe, particularly in certain epidemics. One will see a toxic child with high temperature, respiration up to forty or fifty, and make a diagnosis of pneumonia; the next morning the child will be found almost well, at least as far as the symptoms are concerned.

DR. SCHLUTZ: We have had several cases of acute, severe pneumonia within the last few months and were tremendously impressed with the value of the oxygen chamber in the treatment during the early period of the disease. The anoxemia was relieved, the pulse rate was lowered and the heart action improved. It certainly fits in with the observations of Boothby and Wilder that there is a striking effect, and agrees with their experience in the treatment of the adult.

DR. AMBERG: Oxygen can have a striking effect. Animals gassed with phosgene and put in a chamber with 40 per cent oxygen live and do well while there. Some years ago Knox and I advanced the hypothesis to the effect that any measure which favors oxidation in the tissues is inimical to inflammatory processes. I wonder whether the effect of the oxygen in the chamber is also exercised on the inflammatory process in the lungs, influencing it so that there is less material between the capillaries and the air bearing passages. At the same time the circulation of the lung would be benefited.

DR. NUTTING: These cases in which the patients develop severe symptoms which clear up in one or two days seem to me to be accounted for by the nature of this pathologic process. At first there is congestion, then edema and swelling of the mucous membrane and pouring out of the mucus. If this occurs in the bronchioles, it undoubtedly leads to severe symptoms immediately; likewise, the symptoms can disappear rapidly.

DR. HELMHOLZ: I think this case is a good illustration of how a patient may succumb in spite of the good progress of an actual healing process. The disease led to such intense inflammation that there was complete destruction probably of the greater portion of the epithelial lining of the smaller vessels with resultant exudate. If this child had not been in the oxygen chamber I am convinced that it would not have lived more than three or four days, but owing to the fact that it was in the chamber, life was prolonged. The small amount of tissue left was sufficient to keep the child alive. It was surprising that the child was in such good condition up to the time that death occurred. The bronchi were completely plugged with fibrous tissue, so that there was not a chance of air getting into this portion of the lungs. I think that will explain the resultant atelectasis.

DR. SHANNON: This case brings up an important question. I should like to know how many hospitals have oxygen chambers or adequate facilities for the use of oxygen. I have had to use some makeshift which would not make the treatment worth while. It seems to me that oxygen chambers should be more widely available.

Book Reviews

NURSES, PATIENTS AND POCKETBOOKS. By MAY AYRES BURGESS. Pp. 595, with charts. New York: Published by Committee of Graduate Nursing, 1928.

The grading committee on its observations, affirms that hospitals in the past have graduated, and are still graduating, in increasing numbers, not only too many nurses but too many incompetent nurses. There are many more nurses now in the field than there are available positions, and too many wholly unqualified ones if positions were available. Hospitals have utilized student service, because they have found that student nurses represented a cheap form of labor. They have fitted the student to the financial needs of their institutions, and have utterly failed in giving the student body a thorough and sound education. Women have been admitted to training schools who have had poor educational background and who have been too young to carry the responsibility required of them. All of this is resulting in an inferior product.

Now, however, a general awakening has come. From now on, all efforts must be directed to restricting the number of graduates and at the same time to improving the quality of those who are graduated. From now on, hospitals have no longer any social justification in merely graduating numbers. The demand for numbers has long since been met, indeed far more than met. Emphasis now must be placed on restriction and on education. Only such institutions as are adequately equipped and adequately staffed with a trained teaching corps can henceforth justify their existence. This fact must be brought forcibly to the realization of hospital executives and those in control of training schools. The training of nurses should not be influenced or affected by the financial consideration or difficulties in which hospitals may find themselves.

The sole basis for conducting a training school must be that that institution can furnish such educational experience as is demanded by recognized standards. If an institution finds itself in a position in which it no longer can offer this high type of training, the only alternative left for that institution is to discontinue its school and replace its students by graduates. For hospitals that are qualified to continue to take on students, the increased cost of such service should not be a private affair but should become a public responsibility.

It is at this point that hospitals should do some research work; for in the end, graduate service may be just as cheap as maintaining a training school of high and exacting stadards. If not, the public will find a way to pay for this increasing cost as it finds a way to pay for anything it wants.

By what method can schools reduce their output? Curious as it may seem, the raising of entrance requirements for students does not restrict but rather increases the number of applicants, the desire for higher education being the thing much sought after. It remains, then, for some of the larger schools to take the initiative in the experiment of substituting graduate for student nurses, thereby decreasing their enrollment of new nurses, and at the same time giving employment to women already graduated.

If some of the larger institutions would do this constructive work then at least a beginning would be made in solving this most difficult and perplexing problem. Such, in general, is the theme and trend of thought underlying this illuminating volume of five hundred and ninety-five pages.

SYPHILIS. A TREATISE ON ETIOLOGY, PATHOLOGY, SYMPTOMATOLOGY, DIAGNOSIS, PROPHYLAXIS AND TREATMENT. By HENRY H. HAZEN. Price, $4. Pp. 643, with 165 illustrations, including 16 figures in color. St. Louis: C. V. Mosby Company, 1928.

This work of convenient size contains an amazing amount of material, and presents concisely every aspect of syphilis. It tends to be a compilation, and other

authors have written a few chapters. The text is well illustrated by photographs, photomicrographs and colored plates. Many of the legends preach unforgettable sermons.

The text is valuable in enabling students and practitioners to obtain a bird's-eye view of the disease and become familiar with general principles. The most valuable feature is that of diagnosis, which is incorporated throughout the early chapters and not confined to the one so designated. Syphilis in the negro is discussed.

The size of the text would permit, and its value would be enhanced by inclusion of more of the refinements of syphilologic practice. Individualization of patients for treatment and more detailed study of contraindications to and complications of therapy are of such great practical importance that more space could be devoted to them.

RAD-IMMUNITY. By S. PESKIND. Pp. 6. Cleveland: S. P. Mount Printing Co., 1928.

This is a further development of the author's work on "Systemic and Focal Immunization Through Irradiation," which was reviewed in this journal in May, 1928. In addition, this book contains discussions on the use of rad-immunization in the common types of disorders dependent on focal infection and suggestions for an extension of the uses of this principle.

Directory of Pediatric Societies

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION ON DISEASES OF CHILDREN

Chairman: Dr. W. A. Mulherin, 753 Broad St., Augusta, Ga.
Secretary: Dr. C. A. Aldrich, 545 Lincoln Ave., Winnetka, Ill.
Place: Portland, Ore. Time: July 8-12, 1929.

AMERICAN PEDIATRIC SOCIETY

President: Dr. Henry L. K. Shaw, 142 Washington Ave., Albany, N. Y.
Secretary: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Place: St. Louis. Time: 1929.

THE ASSOCIATION OF AMERICAN TEACHERS OF THE DISEASES OF CHILDREN

President: Dr. Paul W. Emerson, 86 Bay State Road, Boston.
Secretary: Dr. Ralph M. Tyson, 1527 Pine St., Philadelphia.
Place: Portland, Ore. Time: July 8-12, 1929.

AMERICAN CHILD HEALTH ASSOCIATION

President: Hon. Herbert Hoover, 370 Seventh Ave., New York.
Secretary: Dr. Philip Van Ingen, 370 Seventh Ave., New York.

CANADIAN SOCIETY FOR THE STUDY OF DISEASES OF CHILDREN

President: Dr. G. R. Pirie, 20 Madison Ave., Toronto.
Secretary: Dr. A. P. Hart, 1844 Bloor St. West, Toronto.

SECTIONAL

CENTRAL STATES PEDIATRIC SOCIETY

President: Dr. H. T. Price, 121 University Place, Pittsburgh.
Secretary: Dr. A. H. Parmelee, 310 S. Michigan Ave., Chicago.
Place: Milwaukee. Time: Fall, 1929.

SECTION ON PEDIATRICS OF SOUTHERN MEDICAL ASSOCIATION

Chairman: Dr. E. C. Mitchell, 1073 Madison Ave., Memphis, Tenn.
Secretary: Dr. A. J. Waring, De Renne Apts., Savannah, Ga.
Place: Asheville, N. C. Time: November 12-15, 1928.

LOCAL

NORTHERN CALIFORNIA PEDIATRIC SOCIETY

President: Dr. W. L. Channell, Medical Bldg., Oakland, Calif.
Secretary: Dr. J. E. Parrott, Medico-Dental Bldg., San Francisco.
Place: Various Teaching Schools. Time: Monthly.

SOUTHWESTERN PEDIATRIC SOCIETY

President: Dr. Oscar Reiss, Westlake Professional Bldg., Los Angeles.
Secretary: Dr. William Sidney Bowers, 1136 W. 6th St., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

THE ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. W. Wiley Jones, Majestic Bldg., Denver.
Secretary: Dr. Berryman Green, Republic Bldg., Denver.
Place: Children's Hospital, Denver. Time: Third Saturday of each month, 2:30 p. m., September to May, inclusive.

GEORGIA PEDIATRIC SOCIETY

President: W. N. Adkins, Medical Arts Bldg., Atlanta.
Secretary and Treasurer: Dr. E. N. Gleaton, 212 E. Gaston St., Savannah.

PEDIATRIC SECTION OF FULTON COUNTY MEDICAL SOCIETY, ATLANTA, GA.

President: Dr. William W. Anderson, 362 Peachtree St., Atlanta.
Secretary: Dr. Lee Bivings, Exchange Bldg., Atlanta.
Place: Academy of Medicine, 32 Howard St., Atlanta. Time: Second Thursday of each month from October to April, at 8 p. m.

NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. James Herbert Young, 66 Commonwealth Ave., Boston.
Secretary: Dr. Thomas Lanman, 286 Marlborough St., Boston.
Place: Boston Medical Library. Time: Second Friday of each month.

CHICAGO PEDIATRIC SOCIETY

President: Dr. Maurice Blatt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. J. K. Calvin, 104 S. Michigan Ave., Chicago.
Place: City Club. Time: Third Tuesday of each month.

LOUISIANA STATE PEDIATRIC SOCIETY

President: Dr. L. R. De Buys, 1487 Delachaise St., New Orleans.
Secretary-Treasurer: Dr. C. T. Williams, 7401 Burthe St., New Orleans.

DETROIT PEDIATRIC SOCIETY

President: Dr. George E. White, Capitol Theatre Bldg., Windsor, Ontario, Canada.
Secretary: Dr. W. S. O'Donnell, David Whitney Bldg., Detroit.
Place: Children's Hospital of Michigan. Time: No fixed date; approximately once a month.

NORTHWESTERN PEDIATRIC SOCIETY

President: Dr. Ray Shannon, Lowry Bldg., St. Paul.
Secretary: Dr. E. S. Platou, 730 La Salle Bldg., Minneapolis.
Place: University Campus and Rochester. Time: Winter, Spring, Summer, Fall.

ST. LOUIS PEDIATRIC SOCIETY

President: Dr. Park J. White, Beaumont Bldg., St. Louis.
Secretary-Treasurer: Dr. Ralph L. Cook, 508 N. Grand Blvd., St. Louis.
Place: St. Louis Children's Hospital. Time: At least once a year and contingent thereafter on the arrangement of a suitable program.

NEBRASKA-IOWA PEDIATRIC SOCIETY

President: Dr. Floyd Clarke, City Nat'l Bank Bldg., Omaha.
Secretary: Dr. R. B. Eldredge, Medical Arts Bldg., Omaha.
Place: Add-Sell Restaurant. Time: Second Thursday of each month from October to May, inclusive, 6 p. m.

BROOKLYN PEDIATRIC SOCIETY

President: Dr. Louis M. Ruderman, 961 Eastern Parkway, Brooklyn.
Secretary: Dr. Joseph F. Paulonis, 1357 Bushwick Ave., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

BRONX PEDIATRIC SOCIETY

President: Dr. William Hinz, 1964 Grand Concourse, New York.
Secretary: Dr. L. H. Barenberg, 1749 Grand Concourse, New York.
Place: Concourse Plaza, 161st St. and Grand Concourse, New York.
Time: Second Wednesday of each month, except June, July, August and September.

CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various Cities in New York. Time: Fourth Thursday of October, January and April.

PEDIATRIC SECTION OF THE NEW YORK ACADEMY OF MEDICINE

President: Dr. Stafford McLean, 17 East 71st St., New York.
Secretary: Dr. F. Elmer Johnson, 300 West End Ave., New York.
Place: New York Academy of Medicine, 2 East 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

ROCHESTER PEDIATRIC SOCIETY

President: Dr. Paul W. Beaven, 26 S. Goodman St., Rochester, N. Y.
Secretary: Dr. Edwin R. Reynolds, 1620 St. Paul St., Rochester, N. Y.
Place: Arranged by Program Committee. Time: First Friday of each month from October to May.

THE PEDIATRIC SECTION OF THE ACADEMY OF MEDICINE OF CLEVELAND

Chairman: Dr. J. W. Epstein, 3912 Prospect Ave., Cleveland.
Secretary: Dr. Charles W. Burhans, 14805 Detroit Ave., Lakewood, O.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

PHILADELPHIA PEDIATRIC SOCIETY

President: Dr. Edward L. Bauer, Jefferson Medical College, Philadelphia.
Secretary: Dr. John Porter Scott, 2040 Pine St., Philadelphia.
Place: College of Physicians, 19 S. 22nd St., Philadelphia. Time: Second Tuesday of each month from October to June, inclusive.

PITTSBURGH PEDIATRIC SOCIETY

President: Dr. Zaccheus R. Scott, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. Theodore C. Elterich, Highland Bldg., Pittsburgh.
Place: Pittsburgh Acadamy of Medicine. Time: Second Friday, alternate months.

MEMPHIS PEDIATRIC SOCIETY

President: Dr. W. D. Blue, 1024 Madison Ave., Memphis, Tenn.
Secretary: Dr. Gilbert J. Levy, 899 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

TEXAS PEDIATRIC SOCIETY

President: Dr. Edwin G. Schwarz, Medical Arts Bldg., Forth Worth.
Secretary-Treasurer: Dr. Ramsey Moore, Medical Arts Bldg., Dallas.

RICHMOND PEDIATRIC SOCIETY

President: Dr. T. D. Jones, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. W. Ambrose McGee, West End Medical Bldg., 2618 Grove Ave., Richmond, Va.
Place: Westmoreland Club, 601 E. Grace St., Richmond, Va.
Time: First Thursday of each month (other than during the summer).

VIRGINIA PEDIATRIC SOCIETY

President: Dr. Lawrence T. Royster, University.
Secretary: Dr. William B. McIlwaine, Petersburg.

NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. E. D. Carder, 736 Granville St., Vancouver, B. C.
Secretary and Treasurer: Dr. George A. Lamont, 736 Granville St., Vancouver, B. C.

SEATTLE PEDIATRIC SOCIETY

President: Dr. George B. McCulloch, Cobb Bldg., Seattle.
Secretary: Dr. C. L. Smith, Stimson Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, at 7 p. m.

MILWAUKEE PEDIATRIC SOCIETY

President: Dr. A. B. Schwartz, 418 E. North Ave., Milwaukee.
Secretary: Dr. M. G. Peterman, 167 Seventeenth St., Milwaukee.
Place: Children's Hospital, Milwaukee. Time: Second Wednesday of each alternate month, beginning with February.

PEDIATRIC SECTION, ACADEMY OF MEDICINE, TORONTO

President: Dr. M. A. Cox, 34 St. Clair Ave. West, Toronto.
Secretary: Dr. F. R. Hassard, 1361 King St. West, Toronto.
Time: November 17, December 15, February 16 and March 15.

THE AMERICAN JOURNAL OF DISEASES OF CHILDREN is published by the American Medical Association as a means for the advancement of knowledge of the diseases of children and for the publication of scientific investigation in this field.

Manuscripts for publication, books for review, and correspondence relating to the editorial management should be sent to Dr. Clifford G. Grulee, 310 South Michigan Avenue, Chicago, or to any other member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed, AMERICAN JOURNAL OF DISEASES OF CHILDREN, American Medical Association, 535 North Dearborn Street, Chicago.

Articles are accepted for publication on condition that they are contributed solely to the AMERICAN JOURNAL OF DISEASES OF CHILDREN. Manuscripts must be typewritten, preferably double spaced, and the original copy should be submitted. Zinc etchings and halftones of illustrations will be supplied by the Association when the original illustrations warrant.

Footnotes and bibliographies should conform to the style of the Quarterly Cumulative Index Medicus, published by the American Medical Association. This requires, in order given: name of author, title of article, name of periodical, with volume, page, month — day of month if weekly — and year. A complete list of abbreviations for standard periodicals, together with a full discussion of the style of the A. M. A. publications, appears in The Art and Practice of Medical Writing, a comprehensive book on the preparation of medical manuscripts, published by the American Medical Association. Price, $1.50.

Matter appearing in the AMERICAN JOURNAL OF DISEASES OF CHILDREN is covered by copyright; but, as a rule, no objection will be made to its reproduction in reputable medical journals, if proper credit is given. However, the reproduction for commercial purposes of articles appearing in the AMERICAN JOURNAL OF DISEASES OF CHILDREN or in any one of the other publications issued by the American Medical Association will not be permitted.

Authors will receive one hundred reprints free; additional reprints may be obtained at cost.

THE AMERICAN JOURNAL OF DISEASES OF CHILDREN is published monthly. Annual subscription price (two volumes): Domestic, $5.00; Canadian, $5.40; foreign, $5.75, including postage. Single copies, 75 cents, postpaid.

Checks, etc., should be made payable to the AMERICAN MEDICAL ASSOCIATION.

OTHER PERIODICAL PUBLICATIONS
of the American Medical Association

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION—Weekly. Covers all the medical sciences and matters of general medical interest. Illustrated. Annual subscription price (two volumes): Domestic, $5.00; Canadian, $6.50; foreign, $7.50. Single copies, 20 cents.

ARCHIVES OF INTERNAL MEDICINE—Monthly. Devoted to the publication of advanced, original clinical and laboratory investigations in internal medicine. Illustrated. Annual subscription price (two volumes): Domestic, $5.00; Canadian, $5.40; foreign, $5.75. Single copies, 75 cents.

ARCHIVES OF NEUROLOGY AND PSYCHIATRY—Monthly. A medium for the presentation of original articles on nervous and mental diseases, with abstracts from foreign and domestic literature, book reviews, society transactions, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY—Monthly. Devoted to advancing the knowledge of and progress in cutaneous diseases and syphilis. Publishes original contributions and full abstracts of the literature on these two subjects, transactions of the important dermatological societies, book reviews, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

ARCHIVES OF SURGERY—Monthly. Devoted largely to the investigative and clinical phases of surgery, with monthly reviews on orthopedic and urologic surgery. Well illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

ARCHIVES OF OTOLARYNGOLOGY—Monthly. A medium for the presentation of original articles on diseases of the ear, nose and throat, with abstracts from foreign and domestic literature, book reviews, the transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): Domestic, $6.00; Canadian, $6.40; foreign, $6.75. Single copies, 75 cents.

ARCHIVES OF PATHOLOGY—Monthly. A periodical devoted to the publication of original contributions in the field of pathology, with abstracts from foreign and domestic literature, book reviews, the transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): Domestic, $6.00; Canadian, $6.40; foreign, $6.75. Single copies, 75 cents.

QUARTERLY CUMULATIVE INDEX MEDICUS—Quarterly. A complete subject and author index to the worth-while current medical literature of the world. Issued four times a year. Second and fourth volumes bound for permanent reference. Subscription price, calendar year: Domestic, $8.00; Canadian, $8.50; foreign, $8.75.

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn St. CHICAGO

CONTENTS

PAGE

Healing in Infantile Scurvy as Shown by X-Ray. Stafford McLean, M.D., New York, and Rustin McIntosh, M.D., Baltimore 875

The Preventorium Child: A Clinical Review of Three Hundred Cases. I. D. Bronfin, M.D., Denver 931

A Comparison of Cod Liver Oil and Ultraviolet Light for Use in the Prevention of Rickets. Arthur D. Holmes, Ph.D.; Edwin T. Wyman, M.D.; Lawrence W. Smith, M.D., and Madeleine G. Pigott, B.S., Boston 952

An Investigation of the Prenatal Factor in the Susceptibility of Infants to Rickets. Alfred F. Hess, M.D., and Mildred Weinstock, B.S., New York 966

Stimulation of Growth of School Children by Small Supplementary Feedings. Agnes Fay Morgan, Ph.D., and Lydia Warren, B.S., Berkeley, Calif 972

Metabolism of Undernourished Children: VI. Caloric Balance. Chi Che Wang, Ph.D.; Margaret Frank, B.S., and Mildred Kaucher, B.S., Chicago 979

A Clinical Study of Enuresis. Adrien Bleyer, M.D., St. Louis 989

Case Reports:

Sickle Cell Anemia. Martha Wollstein, M.D., and Katherine V. Kreidel, A.B., New York 998

Allergy in an Infant of Three Weeks: Report of a Case and Comment. George M. Lyon, M.D., Huntington, W. Va 1012

Intussusception Due to Papilloma in a Child of Twenty-One Months. Edward J. Lamb, M.D., Santa Barbara, Calif 1017

Progress in Pediatrics:

Sinusitis in Children. Simon L. Ruskin, M.D., New York 1020

Pediatric Biographies:

Felix Platter. John Ruhräh, Baltimore 1037

News and Comment 1040

Abstracts from Current Literature 1041

Society Transactions:

Chicago Pediatric Society 1066

Northwestern Pediatric Society 1079

Book Reviews 1087

Directory of Pediatric Societies 1089